PERIODICAL ROOM
GENERAL LIBRARY
UNIV. OF MICH.
MAY 5 1950

The Journal of HEREDITY

"GENE" FACES THE WORLD
(SEE PAGE 34)

The Decline in Intelligence

Self-Incompatibility in Guayule

Labelling the Blood Group Genes

A "Toe-Binding" Gene in the Fowl

VOLUME 41 FEBRUARY, 1950 NUMBER 2

The American Genetic Association

An incorporated organization devoted to promoting a knowledge of the laws of heredity and their application to the improvement of plants, animals, and human racial stocks

Honorary Emeritus President, DAVID FAIRCHILD

MARCUS RHOADES, President — Vice-President, GEORGE M. DARROW

Honorary Vice-Vresident, W. E. CASTLE

RONALD BAMFORD, Secretary — Treasurer. SAMUEL L. EMSWELLER

Managing Editor, ROBERT C. COOK

Business Manager, VIRGINIA GLEIM — Assistant Editor, BARBARA LAKE

EDITORIAL BOARD

M. DEMEREC — RALPH W. PHILLIPS

H. J. MULLER — LAURENCE H. SNYDER — PAUL POPENOE

Members of the Council are ex-officio members of the Executive Board

COUNCIL

O. S. AAMODT, Head Agronomist, Division of Forage Crops and Diseases, Plant Industry Station, Beltsville, Md.

RONALD BAMFORD, Professor of Botany, University of Maryland, College Park, Md.

GEORGE M. DARROW, Horticulturist in Charge of Small-Fruit Breeding, Horticultural Field Station, Beltsville, Maryland.

HAIG DERMEN, Cytologist, Division of Fruits and Vegetable Crops and Diseases, Plant Industry Station, Beltsville, Maryland.

SAMUEL L. EMSWELLER, Principal Horticulturist, Division of Fruit and Vegetable Crops and Diseases, Plant Industry Station, Beltsville, Md.

DAVID FAIRCHILD, Agricultural Explorer (Retired), U. S. Department of Agriculture.

H. J. MULLER, Professor of Zoology, Indiana University, Bloomington, Indiana.

HUGH C. McPHEE, Assistant Chief of Animal Husbandry Division, U. S. Department of Agriculture, Washington, D. C.

FREDERICK H. OSBORN, Deputy United States Representative on the United Nations Atomic Energy Commission, and on the United Nations Commission for Conventional Armaments.

MARCUS M. RHOADES, Professor of Botany, University of Illinois, Urbana, Illinois.

WILLIAM VOGT, Chief, Conservation Section, Pan American Union, Washington 25, D. C.

SEWALL WRIGHT, Professor of Zoology, University of Chicago.

ADVISORY COMMITTEE

E. B. BABCOCK, Genetics Department, University of California, Berkeley, California.

E. A. BESSEY, Professor of Botany, Michigan State College, Lansing, Michigan.

A. F. BLAKESLEE, Professor of Botany, Smith College, Northampton, Mass.

W. E. BRYAN, Professor of Plant Breeding, University of Arizona, Tucson, Arizona.

RUSSELL W. DUCK, Editor, *The Rural New Yorker*. New York City.

C. M. GOETHE, Sacramento, California.

HARRISON R. HUNT, Professor of Zoology, Michigan State College, East Lansing.

D. F. JONES, Plant Breeder, Connecticut Agricultural Experiment Station, New Haven, Conn.

M. A. JULL, Professor of Poultry Husbandry, University of Maryland, College Park, Md.

C. C. LITTLE, Director, Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine.

JAY L. LUSH, Professor in Animal Husbandry, Iowa State College, Ames, Iowa.

H. H. NEWMAN, Emeritus Professor of Zoology, University of Chicago, (Clearwater, Fla.).

T. S. PALMER, Expert in Game Conservation, U. S. Dept. of Agriculture, Washington, D. C.

JOHN H. PARKER, Director, Midwest Barley Improvement Association, Milwaukee, Wisconsin.

JOHN W. SCOTT, Professor of Zoology, University of Wyoming, Laramie, Wyoming.

A. D. SHAMEL, Physiologist, U. S. Department of Agriculture, Riverside, California.

GEORGE H. SHULL, Professor of Biology, Princeton University, Princeton, New Jersey.

EDMUND W. SINNOTT, Professor of Botany, Yale University, New Haven, Conn.

W. W. SMITH, Professor of Animal Husbandry, Purdue University, Lafayette, Indiana.

A. B. STOUT, Director of Laboratories, New York Botanical Gardens, New York City.

W. T. SWINGLE, Plant Physiologist (Retired), U. S. Department of Agriculture.

W. H. TOMHAVE, American Aberdeen-Angus Breeders' Association, Chicago, Ill.

E. N. WENTWORTH, Armours' Bureau of Agr'l Research and Economics, Chicago, Illinois.

P. W. WHITING, Zoological Laboratory, University of Pennsylvania, Philadelphia, Pa.

The JOURNAL OF HEREDITY is published monthly by the AMERICAN GENETIC ASSOCIATION at 32nd Street and Elm Avenue, Baltimore 11, Md., and 1507 M St., N. W., Washington 5, D. C. Address all communications regarding editorial matters to 1507 M. St., N. W., Washington, D. C. Communications regarding the business management of THE JOURNAL OF HEREDITY may be addressed to the publication office, 32nd Street and Elm Avenue, Baltimore, Md., or to 1507 M St., N. W., Washington 5, D. C. Entered as second-class matter February 24, 1923, at the postoffice at Baltimore, Maryland. Acceptance for mailing at the special rate of postage provided for in section 1103, Act of October 3, 1917 authorized December 29, 1918.

Contents copyrighted 1949 by the American Genetic Association. Unauthorized reproduction of material from THE JOURNAL not permitted. The title "Journal of Heredity" is registered as a trade-mark in the U. S. Patent Office.

$5.00 a Year. 50c a Copy. Prices of back numbers on application.

The

Journal of Heredity

Vol. XLI FEBRUARY, 1950 No. 2

CONTENTS

Syndactylism in the Chicken
By D. C. Warren 31

"Light"—A New Mouse Color
By E. C. MacDowell 35

A Piebald Mutation in Sheep
By P. W. Gregory 39

Hereditary General Gingival Hyperplasia
By Stanley M. Garn and Charles E. Hatch 41

The Inheritance of Flecked and Mark In *Nemesia strumosa*
By Herbert Parkes Riley 43

Self-Incompatibility in Guayule
By D. U. Gerstel and Mary E. Riner 49

Notes and Reviews

"Gene"—The Hybrid Bear 34

A "Group" Theory of Human Origins
By T. D. Stewart 36

An Appraisal of Charles Darwin's Historical Role
By Th. Dobzhansky 40

The Trend of Scottish Intelligence
By D. Grahn 47

Jackson Laboratory Research Facilities for Guest Investigators 48

Blood Group Symbols and Genes
By P. W. Whiting 55

— URGENTLY NEEDED —

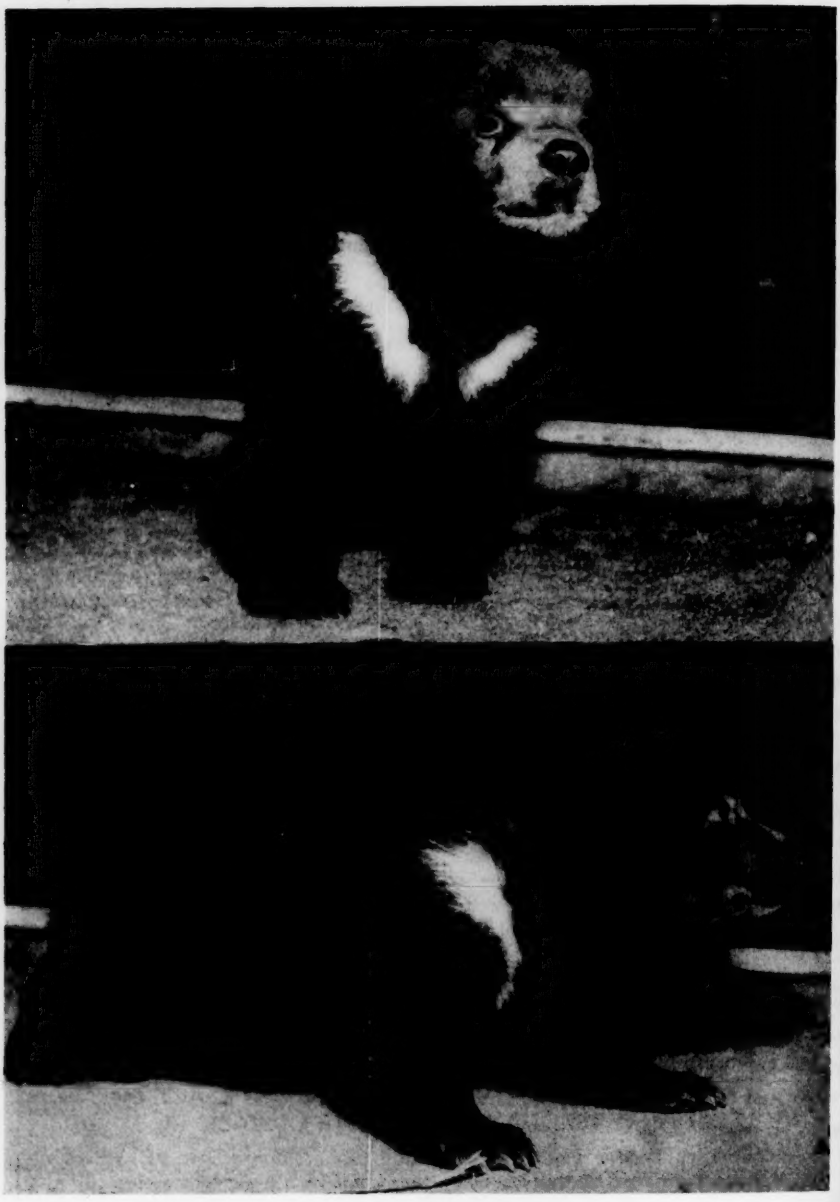
The following two issues of the JOURNAL OF HEREDITY are exhausted:

THESE ISSUES—OCTOBER 1945, FEBRUARY 1947— ARE URGENTLY NEEDED TO COMPLETE LIBRARY FILES.

Members will confer a favor by returning unwanted copies to us so that war-devastated libraries may be furnished with unbroken series.

WE WILL PAY $1.00 PER COPY FOR EACH OF THESE ISSUES OF THE *JOURNAL*.

Mail copies to
AMERICAN GENETIC ASSOCIATION
1507 M. Street, N. W. Washington 5, D. C.



International News Photos

"GENE"—A GENETIC PHENOMENON

Figure 1

The Washington Zoo became aware of a genetical blessed event of considerable importance when Mama F_1, a hybrid Polar $\times$ Kodiac bear, pushed out of her den the lone survivor of a litter of six. This wee F_2, yclept "Gene" by Zoo Director William Mann is prospering and has become the toast of Washington Zoo-goers, drawing a crowd which has emptied even the monkey-house.

SYNDACTYLISM IN THE CHICKEN*

D. C. WARREN
Kansas Agricultural Experiment Station

THIS condition was first brought to the writer's attention in 1940. On repeated occasions since then, flock inspection work in Kansas has brought reports of the instances of this defect in the chicken. The incidence appears to be relatively low, however. A request addressed to the inspectors to be on the lookout for the trait has never brought in more than about six individuals in a year, although the total number of birds handled was in the hundreds of thousands. In all instances the syndactylous birds were of the White Plymouth Rock variety which comprised about one-third of the birds inspected.

The chicken's foot normally shows a narrow web constituted of two layers of skin between and at the base of the toes. This webbing is sufficiently narrow and uniform, however, to make most cases of syndactylism easily identifiable, but occasionally the expression is so slight that it may be overlooked. Instances of syndactylism in one foot only were rarely observed. Webbing varies both in its extent distally and in width between the toes. In extreme cases the two toes are so closely tied together that very little web is visible and the nail is pulled out of its median position. Such birds are handicapped in walking since the foot does not have its normal spread. Other individuals show considerable width of web between the toes but never to the extent found in swimming birds.

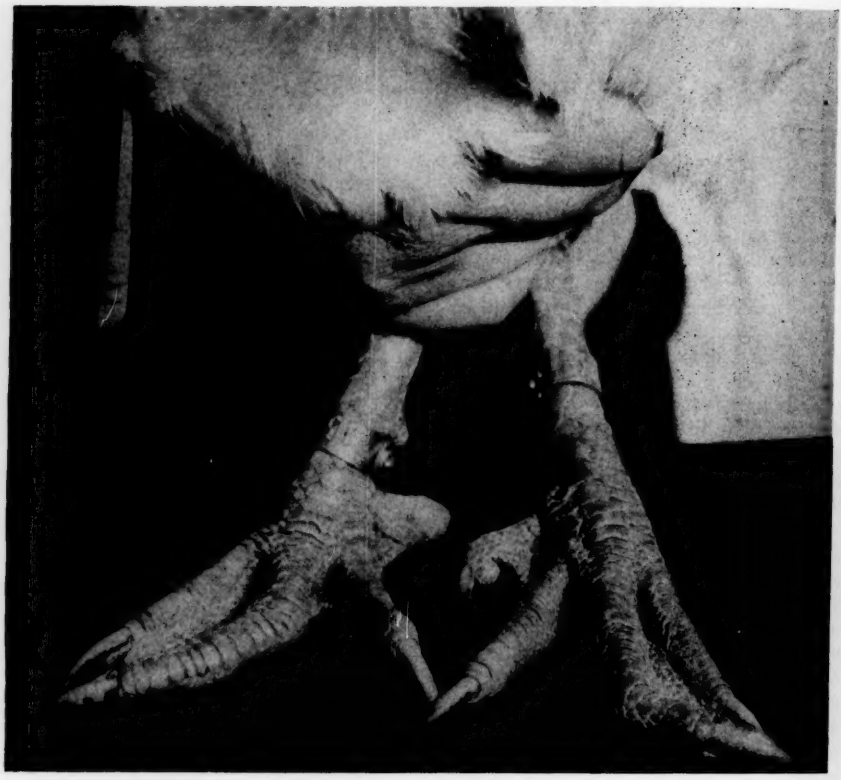
Syndactylism has been described in many animals, including man. In man it usually has behaved as a dominant though not completely so. Digits II and III are more commonly affected in man. Davenport[2] first noted the heritability of syndactylism in the chicken and it was in a Dark Brahma which also has leg feathering. The writer also has observed some instances of syndactylism in breeds with feathered legs. Danforth[1] suggested that syndactyly, brachydactyly, and leg feathering were associated in heredity. It is noteworthy that the syndactylism here reported was in a breed not carrying leg feathering. Davenport listed syndactylism as a dominant but his opinion is based largely on the observation that syndactyls mated with syndactyls failed to breed true for syndactylism.

Inheritance

Although over 4400 birds were involved in crosses of this study of syndactylism, the mode of inheritance remains obscure. There appears to be considerable irregularity in the incidence of the defect in matings of the same type.

In Table I are shown results of mating syndactyls to normals. The syndactyls included in these matings were usually segregates from backcrosses of F_1 individuals to syndactyls or from matings of syndactyls by syndactlys, since it was not possible to obtain a true breeding flock of syndactyls. It is seen that the numbers of syndactyls resulting from these various matings of syndactylous with normal birds are quite variable. The first two matings listed in Table I would indicate almost complete recessiveness of syndactylism, while the other matings showed a high incidence of syndactyls. It is noted that the first two matings are reciprocals of the remaining ones and the difference might be attributed to sex linkage. The difference in results cannot be accounted for on this basis since the syndactyls appear in about the same proportion in both males and females. If sex-linkage were involved then the syndactyls should have been females. It would seem then that

*Contribution No. 183, Department of Poultry Husbandry.



"BOUND" TOES

Figure 2

A case of syndactylism in the chicken where the distal extension is so extreme that both the median and outside toes are pulled out of their normal positions.

the variability must be due to differences in the genetic constitution of either the syndactyls or the normal stocks.

The data in Table I suggest that the choice of normal stocks may influence the incidence of syndactyls in the F_1 generation. The same syndactylous male A (third and fourth matings Table I) when mated with White Wyandotte and with White Leghorn females produced offspring with differing proportions of syndactyls. In the first two matings several syndactyls served as the maternal parents and only one syndactylous offspring resulted among 54. In comparison with the matings when the sire was the syndactyl, chance should have given more syndactyls in the first two matings if the different ratios of syndactyls were chargeable to the varying genetic constitution of the syndactylous parent. It would seem then that various normal parent stocks used, differed with regard to some genetic factor which influenced the dominance of syndactylism, or that they carried the gene for syndactylism. It is of interest that one of the parent stocks, the Light Brahma, carries leg feathering which earlier workers be-

lieved was associated with syndactylism, but in this cross no syndactyls appeared in the F_1 generation. It was the White Wyandotte and White Leghorn matings which threw considerable numbers of syndactyls in the F_1 generation, but neither of these breeds was known ever to have thrown any syndactyls although the White Leghorns were observed in large numbers.

Anomalous Behavior of F_1

A question of interest is whether the syndactyls appearing in the F_1 generation differ genotypically from the normals in the same mating. In Table II a comparison is made of the relative ability of F_1 generation syndactyls and normals to transmit syndactylism. The first comparison in Table II is of a syndactylous male and a normal male from the F_1 generation when mated with White Leghorn females, and a wide difference in behavior is noted. The offspring of the syndactylous F_1 male were 81 syndactyls and 209 normals, while the offspring of the normal F_1 male were all normal. In the last two comparisons (Table II) where both F_1 generation males and females were compared and mated with syndactyls, instead of normal Leghorns, there was again evidence for differences in the genotype of the syndactyls and normals occurring in the F_1 generation. The results were similar whether the comparison was of F_1 males or of F_1 females, and in each case the syndactyls produced more syndactylous offspring than normal offspring, while the reverse was true if F_1 generation normals were used. The results in Table II indicated that the normals and syndactyls appearing in the F_1 generation are actually of a different genetic constitution rather than the result of some factor influencing the dominance of syndactylism.

Several matings were made of syndactyls by syndactyls, each having segregated from backcrosses of F_1 individuals to syndactyls. The totals from the four matings of syndactyls were 166 syndactyls to 84 normals. In each case the matings were headed by a single male. These matings differed from those in the third and fifth lines of Table II only in that in the latter matings one syndactylous parent was from F_1 generation. In each case the matings of syndactyls by syndactyls produced offspring about two-thirds of which were syndactyls. This would indicate that the F_1 generation syndactyls had the same genotype as those from the back cross generation. None of the four males heading the matings of syndactyls by syndactyls which produced a total of 166 syndactyls to 84 normals, produced only syndactyls but in each mating the syndactyls exceeded the normals. Another mating of F_1 normal males by F_1 normal females produced an F_2 generation which resulted in 4 syndactyls to 32 normals. The proportion of syndactyls to normals is much too low to provide the simple interpretation of syndactylism as a recessive trait. The first two matings in Table I would seem to place syndactylism in the recessive group of characters but the results of other matings in this table make this interpretation unacceptable.

The results in Table II where both syndactylous and normal F_1 individuals were outcrossed to White Leghorns might be interpreted to mean that more than one pair of factors are involved and that certain combinations of such factors produce syndactylism even when they are in a heterozygous state. The data in the last four matings of Table II are actually backcross data and none of the results permit any simple explanation of the inheritance of syndactylism. It should be noted that two other factors in the chicken, ungual osteodystrophy[5] and double spurs[3], each though behaving as recessives, occasionally threw the recessive trait in the F_1 generation.

TABLE I.—Matings of Syndactyls *(S)* with Normals *(N)*

	Females		Males		Totals	
	S	N	S	N	S	N
Crossbred male by syndactylous female	1	26	—	11	1	37
Light Brahma male by syndactylous female	—	5	—	11	—	16
White Wyandotte female by syndactylous male *A*	—	—	—	—	15	14*
White Leghorn female by syndactylous male *A*	9	3	5	3	14	6
White Leghorn female by syndactylous male *B*	30	52	38	66	68	118
TOTALS	40	86	43	91	98	191

*Sex not determined.

Syndactylism Possibly Related to Polydactylism

Attention is directed to data published by the writer[4] on the relationships of syndactylism with two types of polydactylism. The matings were designed to determine whether these two types of polydactylism were in the same linkage group with syndactylism. Polydactyls and syndactyls were crossed and the resulting F_1 generation offspring were backcrossed to syndactylous, non-polydactylous individuals. Regardless of whether syndactylism and polydactylism

TABLE II.—Comparison of the Genotypes of Syndactylous *(S)* and Normal *(N)* Offspring Appearing in the Same Matings in the F_1 Generation.

Type of Mating	Offspring S	Offspring N
F_1 Syndactylous male by White Leghorn females	81	209
F_1 Normal male by White Leghorn females	—	107
F_1 Syndactylous male by syndactylous females	80	68
F_1 Normal male by syndactylous females	809	1615
F_1 Syndactylous females by syndactylous male	67	36
F_1 Normal females by syndactylous male	372	759

come into the cross in the same parent or from opposite parental sides, there was a tendency for syndactylism and polydactylism to show repulsion in segregation. The syndactylous, polydactylous class was small. Since polydactylism and syndactylism each show low penetrance, this discrepancy would be expected to some degree, but the shortage was greater than could be accounted for on this basis. From one test involving the duplicate type of polydactylism the segregating groups were 271 duplicate non-syndactyl, 150 non-duplicate syndactyl; 144 non-duplicate non-syndactyl, and 4 duplicate syndactyl. The duplicate-syndactylous group is so small that it would appear that there may be some sort of inhibitory action between syndactylism and polydactylism. The exact nature of this action is not clear.

Summary

The data on syndactylism in the chicken will permit no simple explanation of its mode of inheritance. The results in the F_1 generation matings varied from zero to almost 50 percent syndactyls. No matings of syndactyls to syndactyls or syndactyls to heterozygotes produced only syndactylous offspring. Thus this case of syndactylism in the White Plymouth Rock breed seems to be due to a somewhat complicated genetic constitution. The wide variance in the F_1 generation results suggests that breeds not showing syndactylism may carry factors influencing its expression. The syndactylism here reported is not associated with leg feathering. There is no evidence of sex-linked factors being involved.

Literature Cited

1. Danforth, C. N. *Amer. Jour. Anat.* 25:97. 1919.
2. Davenport, C. B. Carnegie Inst. Wash. Pub. 121. 1909.
3. Warren, D. C. *Jour. Hered.* 37: 323-324. 1946.
4. ———. *Genetics* 34:333-350. 1949.
5. ———, C. D. Mueller, and F. B. Hutt. *Jour. Hered.* 35: 354-358. 1944.

◆

"GENE"—THE HYBRID BEAR

THE second half century of genetics may see the development of a new specialty, ursine genetics, if recent events at the Washington Zoo represent a trend. For several years Dr. William Mann has proudly exhibited his hybrid bears—the progeny of a cross between the Kodiac brown bear of Alaska and the polar bear. These creatures, of imposing size and tawny yellow color reminiscent of a Palomino horse, have been mated several times. The resulting progeny have only briefly survived.

Since bear cubs are born during the winter hibernation period, the early weeks of a bear cub's life is shrouded in considerable mystery. The time of birth can be determined quite accurately for the keepers can hear the rather noisy suckling of the new-born infants. But the process of development remains hidden until the mother bear brings the cubs out of the den.

In the winter of 1949-50 litters of three were born to two of Dr. Mann's F_1 females. Of the litter born on January 2, 1950, the mother nursed the young for little more than a week, and then expelled one lone survivor.

Little "Gene's" life hung in the balance for several days after he was thus unceremoniously cast out into the world. At an age of three months he is making excellent progress. Apparently he is as strong as a normal bear cub of his age. He is far and away the "feature presentation" of the Washington Zoo in the spring of 1950.

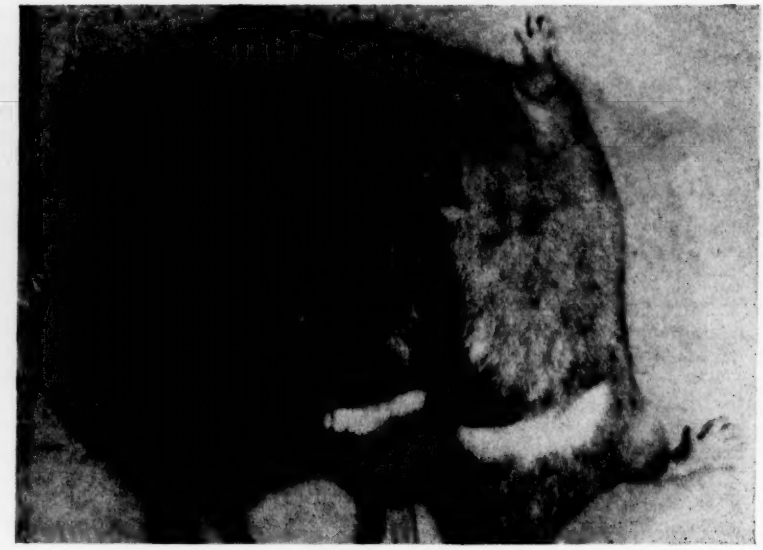
It is unlikely that bears will replace mice, rats, and guinea pigs as subjects for genetic experimentation. The significance of the interfertility of the two arctic zone species is hard to appraise since so little has been done in ursine genetics. Among mammals we find a wide variation in the fertility of hybrids. Among the Equidae the mule is almost invariably sterile, as are zebra-ass hybrids. The bovidiae show a different situation. There, in rather wide crosses between cattle, bison, and yak, we find complete, or considerable, fertility. Such considerations permit geneticists to contain their astonishment at the survival of this most interesting hybrid.

It is to be hoped that little Gene is only a harbinger of a large crop of siblings or cousins in the years to come. Will they show, as might be expected in the F_2 generation of such a cross, brown, white, and khaki coloration? Will some of them take to the sea, and will others cleave to the land? We can hardly *bear* to wait for time to give us the answers!

—R. C.

"LIGHT"--A NEW MOUSE COLOR

E. C. MacDowell
Department of Genetics, Carnegie Institution of Washington, Cold Spring Harbor, N. Y.



THREE GRADES

Figure 3

Black, dark (L^tL^t) and *light* (L^tL^t). Clipped patches show base of hair which actually is very light gray on the *dark* mouse and virtually white on the *light* mouse.

IN the 76th generation of sib-matings in the black strain C58, a hair-color mutation appeared that is epistatic and closely linked, if not allelomophic, to black. The proposed designation of the locus is *light* L^tL^t (B^l if allelomorphism in the B series should be established). The homozygotes are *hair brown* (Ridgway) at the hair tips and the rest almost white. These are called *light* in contrast to the heterozygotes, which are called *dark*, for the tips in L^t + are darker, *chaetura drab* (Ridgway), and the pigment extends further down the hair and the lower part is very light gray. (Figure 3). Homozygotes and heterzygotes are distinguishable even in the second week when only the hair tips are showing. As the hair lengthens, the general effect is rapidly lightened, and this lightening is continued even after full size is attained. Moulting patterns occasionally appear. Eye color shows no obvious deviation from black.

Albinism and agouti segregate independently of L^t. Agouti combines with L^tL^t and L^t + just as it does with black and brown, with no modification of the red band, which virtually conceals the pigment of the hair tips but leaves the light base to identify L^t. Thus in the presence of agouti, L^t can not be recognized certainly until the hair is well grown and the distinction between L^tL^t and L^t+ is very much less conspicuous than in non-agouti animals.

Table I gives the matings within strain C58 (BB), which indicate the monogenic relations between L^tL^t, L^t+, and ++. L^tL^t crossed with brown (strain

Daab) gave F_1 all dark, indistinguishable from the *darks* of Table I. F_2 of this cross (Table II) segregated for *light*, *dark* and brown (1:2:1) but no blacks appeared, nor any suggestion of brown varieties of L^tL^t, and L^t+. Another cross with brown, this time including also albinism and agouti (*Balb*) gave similar results in F_2 (Table III). Black and black agouti were entirely absent and L^tL^t and L^t+, whether combined with agouti or not, gave no recognizable brown classes. Backcrosses of this F_1 to the *bbAAcc* parent (Table IV) gave no black agouti and only one class of *dark* agoutis.

Thus in a total of 88 ++ mice in the F_2 of the two crosses, all were brown instead of 66 blacks and 22 browns. In the backcrosses, 52 ++ mice were all brown instead of 26 blacks and 26 browns. In the light of this evidence of linkage between L^t, and *B*, the failure to observe brown (*bb*) classes of L^tL^t and L^t+ may be interpreted as secondary evidence of the same linkage. Thus in a total of 294 F_2 carrying L^t no *bb* mouse was found instead of 73; and in a total of 60 BC mice carrying L^t no *bb* mouse appeared, instead of 30.

TABLE I.—Matings within strain of origin (C58, *BB*)

Parents	Offspring		
	Light L^tL^t	Dark L^t+	Blk ++
Light × *light*	27	—	—
Light × black	—	52	—
Dark × black		52	61
Dark × *light*	27	24	—
Dark × *dark*	28	32	23

TABLE II.—F_2 *Light* × Brown (L^tL^t *BB* × ++bb, Strain *Daab*.)

Color	Expected independent		Observed	Expected linked
Dark	L^tL^tB	19.7	24	26.2 *LB LB*
	L^tL^tbb	6.5		0
	L^t+B	39.4	61	52.5 *LB +b*
	L^t+bb	13.1		0
Black	++B	19.7		0
Brown	++bb	6.5	20	26.2 *+b+b*

TABLE III.—F_2 of a Cross, *Light* × Balb (L^tL^tCC-*BBaa* × ++*ccbbAA*)

Color	Expected independent		Observed	Expected linked
Light agouti	L^tL^tBA—	38.7	50	51.6 *LB LB A*
	L^tL^tbbA—	12.9		0
Dark agouti	L^t+BA—	77.4	111	103.2 *LB +b A*
	L^t+bbA—	25.8		0
Light	L^tL^tB—	12.9	12	17.2 *LB LB*
	L^tL^tbb	4.3		0
Dark	L^t+B—	25.8	36	34.4 *LB +b*
	L^t+bb	8.6		0
Black agouti	++BA	38.7		0
Brown agouti	++bbA	12.9	54	51.6 *+b +b A*
Black	++B	12.9		0
Brown	++bb	4.3	14	17.2 *+b +b*
Albino	cc	91.7	90	91.7

TABLE IV.—Backcross to Balb (L^t +*Bb*, *Ac*, *Cc*, × ++*bbAAcc*)

Color	Expected independent		Observed	Expected linked
Dark agouti	L^t+BA	24.6	60	49.2 *LB +bA*
	L^t+bA	24.6		0
Black agouti	++BA	24.6		0
Brown agouti	++bA	24.6	52	49.2 *+b +bA*
Albino	cc	98.5	85	98.5 cc

A "GROUP" THEORY OF HUMAN ORIGINS

FEW anthropologists have had opportunities equal to those of Sir Arthur Keith for studying the evidence of human evolution. From the vantage point of the Museum of the Royal College of Surgeons, of which in 1908 he was placed in charge, he has been able to study each new find of fossil ape or man. His well-known books summarizing these discoveries (*The Antiquity of Man*, 1915; *New Discoveries Relating to the Antiquity of Man*, 1931) are standard reference works. Yet to Sir Arthur the material that they present has been but "grist to the mill"; namely, for working out the "machinery of evolution" or "the means and manner by which man had made his ascent." In his own words: "These forty years I have been standing, as it were, at the receipt of custom and, while pursuing my own inquiries, have gathered into my portfolios each fact or idea as it came along in the hope of gaining materials from which to fashion a more precise theory of man's evolution."

From these portfolios, Keith fashioned a volume of *Essays on Human Evolu-*

tion in 1946. Now, in his 84th year he has published a second book of essays (but not so called) explaining in detail what he calls his "group" theory of evolution.* With those who object to entitling this theory "new," on the grounds that "it is simply Darwin's theory extended, modified, and brought up to date," Keith has no quarrel. He says modestly that "at least it is a new rendering of the Darwinian theory."

Unlike Darwin's *Descent of Man,* Keith's theory was not withheld from public view until it had been fully developed. Parts of the "new" theory were announced from time to time over the years. Here they are only assembled and amplified. Critics even have had their say before. And now at least some of their objections are taken into account.

The essential postulate of Keith's theory holds that throughout the entire period of man's major evolution—that is, right down to the dawn of civilization—mankind was divided into isolated local communities, and that these competitive, inbreeding groups—his so-called "evolutionary units"—"provided the most favorable circumstances for bringing about rapid changes in brain and body."

A secondary postulate holds "the 'machinery of isolation' to be resident in the mentality of ape and man." Abetting the tendency to group isolation are certain mental traits characteristic of man and at least incipient in his nearest primate relatives. Among these traits are: a sense of ownership of territory, group spirit, patriotism, cooperation and competition, mental bias, resentment and revenge, desire for status, leadership and loyalty, etc.

Added to all this is the idea that "throughout the later stages of human evolution the tendency has always been towards the production of larger and more powerful evolutionary units"—local groups, tribes, nations.

On this foundation Keith proceeds (1) to explain why the evolutionary fate of man differs so greatly from that of this co-descendant, the chimpaneze; (2) to determine the frontier between apedom and man-dom; (3) to locate the cradle of mankind; (4) to spread man's ancestors abroad; and (5) to explain the distribution of the present racial types.

His answer to the first of these problems, unfortunately, is not an explanation but only the description of an end result:

> To the pre-human groups fell that set of genes which were biased towards making the body and brain dependent on the lower limbs for support and progression, and to deprive the hands and arms of their locomotory function and make them the domestic servants of body and brain. The pre-chimpanzee groups were less fortunate in the "draw" for genes. (p. 168)

As for the second problem, the distinction that Keith would set between fossil ape and fossil man, seems altogether inadequate:

> I would say that any group of the great Primates which has attained a *mean brain volume* of 750 c.c. and over should no longer be regarded as anthropoid, but as human. (p. 206)

His location of the cradle of mankind —problem 3—has arguments in its favor, if as yet limited evidence:

> Where are we to pitch the centre of dispersal? The evidence, as it stands to-day, favours Africa. It is in that continent we find the living anthropoids which are most akin to man in structure of body and of brain; it is there, too, that ground forms of anthropoids lived; the oldest and most primitive of orthograde forms lived in the lower valley of the Nile. If we may select one region as more likely than another, then our choice falls on the uplands of Uganda and Kenya; during Upper Miocene times this area was the home of numerous anthropoids, one of which was akin to the gorilla and chimpanzee and yet in certain features more human than either. (p. 214)

In his efforts to solve problem 4 and get man's ancestors out of Africa, Keith resorts to some strong measures, as for example:

> So far it has been assumed that the pioneer groups were made up of individuals conforming to one type—namely, that of the South African anthropoids. This may not have been the case—there may have been more than one type. Seeing the close relationship of the

*Keith, Sir Arthur. *A New Theory of Human Evolution.* Pp. 451. Price $4.75. Philosophical Library, New York, 1949.

orang to the African chimpanzee and gorilla, it is probable that this anthropoid, too, is of African origin. If this were the case, then it is possible that among the early forerunners of mankind in Africa some had inherited the orang form of skull and forehead. This is what I am assuming. This modification of my theory involves two other assumptions: (1) that it was the orangoid forms that turned westwards into Europe and ultimately reached England, where their further evolution continued; (2) that those characters of the human skull we count modern, such as the mastoid process and chin, have been evolved independently in several races of mankind. (pp. 231-232)

Lastly, his explanation of the fifth problem involves an exaggerated regional homogeneity of present racial types and also an unlikely simple history. Example:

I look upon the aborigines of [Australia, New Guinea, Melanesia and Tasmania] as descendants of a common ancestral stock, their racial divergence being the result of long separation (from mid-Pleistocene times or earlier); the evolutionary changes are such as ensue in populations which are long isolated and inbred. (p. 242) [Incidentally, the Polynesians nowhere are accounted for.]

Beginning with Essay 26, Keith is concerned mainly with the genesis of modern nations and their evolutionary role in race formation. This part requires careful reading because Sir Arthur is unorthodox in using the word race in what he regards as its original sense; that is, "to designate a group or a people involved in the process of differentiation." A nation also fits this definition and hence becomes synonymous with race. This leads to the characterization of such peoples as the Welsh and Jews both as nations and as races. Sir Arthur is unorthodox also in holding that the primary marks of race are psychological (p. 377).

In spite of all the ingenious argument in which the "group" theory is set, it suffers from fixation on a single factor in the intricate process of human evolution—isolation. Other factors, and especially environmental selection, get very little consideration. Yet, on the other hand, genetics supplies the author with an inexhaustible bank of genes and ready mutations to help out whenever the theory fails to account for man's known differentiation and distribution.

Another weakness of the argument is the insistence upon mankind's awareness of his evolutionary potentialities or destiny. The following examples illustrate this:

. . . a group . . . separated from other groups in order to inbreed, and so to work out the evolutionary potentialities of its genes. . . . (p. 41)

. . . human nature . . . has been so evolved as to govern the evolutionary destiny of human groups. (p. 95)

. . . the inhabitants of Ireland . . . set out . . . to exploit the potentialities of their minds and bodies . . . and so bring into existence a distinctive Irish race. (p. 371)

. . . man is a racial-minded animal. (p. 406)

Obviously it is possible to pick flaws in Keith's argument, as well as in his interpretation of the events in human prehistory, but this does not destroy the value of his "group" theory. Indeed, this "new rendering of the Darwinian theory" contains much to recommend it, not the least being the author's clear style of writing.

T. D. Stewart

U. S. National Museum

Genetics News From Japan

It is our most pleasant news to you that the National Institute of Genetics of Japan was established on June 1. The institute was placed at Mishima, about three hours by train from Tokyo. My teacher, Dr. K. Oguma, was appointed as the first director of the Institute. The main staff includes: Dr. Y. Tanaka (silkworm geneticist), Dr. H. Kihara (wheat cytologist), Dr. T. Komai (human and Drosophila geneticist), Dr. Y. Sinoto (plant cytologist), etc.

On October 20-22, we had the Annual Meeting of the Japanese Society of Genetics at Nagoya University, where about 120 articles were presented by geneticists and cytologists. On October 30, the Chromosomics Society of Japan was started at Tokyo, with the support of many specialists. The quarterly journal, *La Kromosomo*, will be published as its organ.

Sajiro Makino

A PIEBALD MUTATION IN SHEEP

And a Suggested Interpretation*

CALICO SHEEP
Figure 4

The mutant piebald ewe lamb, her male black twin, and their mother. The appearance of a spotted pattern in sheep is so unusual as to suggest a number of alternative possibilities.

ROBERTS[1] reported the results of crosses of spotted with self black color in sheep. His data were limited, but the results justified his conclusion that the spotting in question was conditioned by an autosomal recessive gene. Recently, Mr. Benny Paradiso of San Carlos, California, brought to my attention a piebald lamb that occurred on his farm. Black or brown sheep are not uncommon but spotting of the type shown by this lamb is so rare as to be practically unknown to veteran sheep herders or sheep breeders. The origin of this mutant is such that a recessive gene is unlikely. Since this mutant throws some light on coat color inheritance and factor interaction in sheep the object of this note is to place the case on record.

A review of the literature of the genetics of coat color, and of factor interaction in sheep is not needed here, although it is necessary to understand the factors involved and their interactions to produce the phenotypes under consideration. Roberts and White[3] presented convincing evidence that the common type of white coat color is conditioned by a dominant gene which acts as an inhibitor to color. This inhibitor is epistatic, and masks other genes that produce color and color patterns.

When the color inhibitor (*I*) is absent the coat may be either black or brown; however the genetic relationship of black and brown has not been determined. According to Roberts and White[3] one pair of alleles, designated as *B* and *b*, may condition these two colors, the black being dominant, as in most mammals. Another possibility is that the black locus has never mutated and all sheep are homozygous for *B*, the brown color being produced by a genetic or an environmental modification of the action of gene *B*. If the modification is genetic, at least one pair of alleles which modify black to brown belongs in the genetic complex. Roberts also presented data which indicated that more than one genotype produces black. When black individuals of the Black Welch Mountain breed were crossed with ordinary white (*I*) sheep, all the progeny were black, and an F_2 population from such ewes had more black than white individuals, although the proportions in the F_2 did not fit a monohybrid Mendelian pattern. Roberts and White logically suggest that this second type of black is brought about by a factor that in its turn is epistatic to the dominant epistatic inhibitor (*I*). Several types of gene relationships are possible. This second type of black

*This study was supported by the Kellogg Fund of the University of California for the application of Genetics to Farm Mammals.

is rare in breeds of British and Spanish origin, but is found in the Karakul and Asiatic breeds. The piebald mutant (Figure 4) lacks the color inhibitor *I*, and all the blacks involved are of the genotype *ii*.

The Piebald Mutant

The California piebald mutant is a ewe, born twin to a black male. The mother and twins are shown in Figure 4. The white areas on the spotted lamb are large and extend to all parts of the body. The spotting does not resemble any of the recessive piebald mutants shown in Robert's[1] Plates 10 and 11. It rather closely resembles the dominant spotting of horses commonly referred to as piebald, skewbald, or calico.

As yet the mutant has not been used in any test matings and whether or not any such data will be available is problematical. If the parents are of the genotypes logically expected, and the piebald mutant is conditioned by a dominant gene, the factor interactions of the genes involved can be predicted. Although they are not eligible for registration, the parents of the mutant are of Rambouillett breeding. Two assumptions that most animal geneticists would agree upon are: (1) that the white and black coat colors involved in the stock are conditioned solely by the inhibitor alleles *I* and *i;* and (2) that all animals are homozygous for *B*. The piebald mutant is subject to two interpretations; (1) that it is of a recessive type, self color being dominant as in the spotting type studied by Roberts[1]; and (2) that the spotting is conditioned by a dominant gene and is similar to dominant piebald of the horse.[4] The nature of the spotting is such that the second alternative is favored. If the piebald mutation is dominant it was not carried by the black sire of the mutant female although it could have occurred in his germ tract. Since the white dam carries the inhibitor *I*, she may be heterozygous for the piebald condition, or she may have produced a mutant egg. A recessive spotting would have involved both parents.

When all contingencies are considered a dominant spotting gene seems the more probable and the factor interactions have been discussed.

Literature Cited

1. Roberts, J. A. F. *Jour. Genet.* 17:77-83. 1926.
2. ———. *Jour. Genet.* 22:181-190 1930.
3. ——— and R. G. White. *Jour Genet.* 22:165-180. 1930.
4. Wright, Sewall. *Jour. Hered.* 8:561-564 1917.

P. W. Gregory

Universtiy of California
Davis, California

✦

AN APPRAISAL OF CHARLES DARWIN'S HISTORICAL ROLE

IT is useful from time to time to re-examine and re-evaluate the mainsprings of our civilization, particularly during the period of eery calm of political and intellectual reaction in which we have become submerged after the storm of the Second World War. This seems to be the purpose of the Twentieth Century Library, to which Professor P. B. Sears has contributed a compact but exceedingly well written book on Darwin.* Darwin's followers and foes are indeed surprisingly unanimous in regarding him as one of the creators of the modern intellectual climate.

The book is neither a full-dress biography of Darwin, nor an exposition of Darwin's theories, except in so far as it is necessary to explain the historical importance of Darwin's ideas. The modern developments of evolutionism are scarcely mentioned. Professor Sears has written a study of the impact of Darwin's work on the currents of thought, chiefly in the English-speaking countries. Darwin's principal contributions are stated thus: "First and foremost, Darwin advanced, by example, the right of free inquiry and discussion, so long as it goes hand in hand with a scrupulous examination of evidence. Second, he advanced the concept of process, not of being, but of becoming. Far and above everything else that Darwin means, these two principles stand forth clear and uncompromising." And: "Man is facing, in intensified form, two ancient problems—that of living with himself and that of making peace with nature. We call them two problems. Actually we should see them as facets of the same great problem, if we accept Darwin's view that man is a part of nature."

Darwin's influence has helped to free man from spiritual bondage. To those nurtured in admiration of Darwin as one of the greatest scientists who ever lived, this is a familiar truth. But one must beware of a false perspective: there is no unanimity about the historical meaning of Darwin even if one disregards the anti-evolutionists. Sears' book may be considered an answer to a very different evaluation of Darwin's role in the history of ideas published not so long ago by Jacques

**Charles Darwin. The Naturalist as a Cultural Force.* P. B. Sears. Twentieth Century Library. Charles Scribner, New York & London. 124 pp. 1950. $2.00.

Barzun,* one of the most brilliant students of our cultural heritage (albeit Barzun's name is not mentioned in Sears' book). According to Barzun, Darwin is one of those who are responsible for the dreary mechanistic slant of modern thought.

Some readers may feel that Professor Sears has devoted much space to the role of Darwin in the ideological currents of the nineteenth century at the expense of the issues of our own time. The tragic delusion of the so-called "social Darwinism" is dealt with only in passing, as is the problem of eugenics and euthenics. The involvement of Darwinism in the Fascist and Marxist philosophies are described fairly but too briefly. Again one must not forget that what to some of us may seem closed issues and settled problems are still vitally important to many people in many parts of the United States and elsewhere. The book is a very useful and effective statement of the import of Darwin's ideas.

THEODOSIUS DOBZHANSKY
Columbia Universtiy

**Darwin, Marx, and Wagner.* Critique of a Heritage. JACQUES BARZUN. Little ,Brown & Co. New York. 420pp. 1941.

✦

HEREDITARY GENERAL GINGIVAL HYPERPLASIA

STANLEY M. GARN* AND CHARLES E. HATCH†
Forsyth Dental Infirmary for Children, Boston

GUM OVERGROWTH

Figure 5

For three (possibly four) generations some individuals in this family have suffered from excess growth of the gums, which covers nearly all of the teeth. Surgical removal of the excess tissue brings only temporary relief.

GENERAL gingival hyperplasia is a condition marked by extensive overgrowth of the gingivae. It may be caused by localized infection, blood dyscrasia, specific medication, or much more rarely it may be hereditary. In this latter case the overgrowth usually appears by the sixth year of life, and it may interfere with mastication and with occlusion. Attempts to reduce the gingivae surgically have in general been unsuccessful, the overgrowth returning within a few years.

This anomaly has been reported from several states, the British Isles, and from the European Continent. Stones, of England, has noted general gingival hyperplasia in two female siblings.[4] Weski has reported gingival

*Research Fellow in Anthropology, Forsyth Dental Infirmary for Children; Instructor in Anthropology, Harvard University.

†Chief of the Dental Clinic, Forsyth Dental Infirmary for Children.

overgrowth in five generations of a German family.[6] Ruggles in Ohio has treated three of nine members of a Welsh family with a history of gingival hyperplasia in three generations.[3] Other recent reports include Buchner's three cases from Illinois, a mother and her son and daughter,[1] cases studied by Prinz and Greenbaum[2] and mentioned by Thoma.[5] The hereditary nature of this extremely rare anomaly has been stressed since it was first reported in 1878. The evidence indicates that it is not sex linked, and that the gene responsible for the condition is not confined to any one sub-group of the white race, nor is it restricted to any small geographic area.

Case Presentation

A seven year old, healthy, mesomorphic girl of Italian extraction was referred to the Child Growth Clinic of the Forsyth Dental Infirmary because her gingivae nearly covered the crowns of the teeth, leaving only the incisal edges visible (Figure 5). The mother, who is a very reliable informant, was questioned about the existence of this anomaly in the family. She reported that the proband had an affected brother, and several of her own siblings were affected; she herself had been treated unsuccessfully by a plastic surgeon. The occurrence of this hyperplasia in the proband's family is shown in the pedigree (Figure 6). It should be noted that the sister denoted by an asterisk is, at thirteen months of age, too young for us to determine whether the condition is likely to appear. Similarly, presence of gingival overgrowth in the proband's great-grandmother (Figure 6) is problematical, though the mother thinks it was present.

Discussion

This pedigree strongly suggests that we are dealing with a Mendelian dominant, since about half of the siblings are affected in each generation, and the condition extends through three and possibly four generations. Both males and females are affected, and the gingival overgrowth may be transmitted by either males or females in this family line. The ratio of affected to unaffected siblings in three generations does not differ significantly from the expected 1:1 ratio. Thus the anomaly appears to be inherited as a simple autosomal dominant with high penetrance in this pedigree.

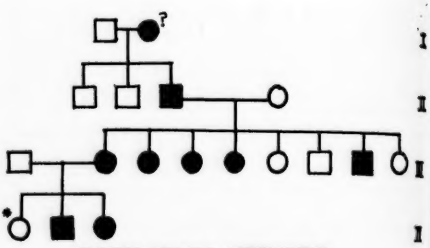


Figure 6

In view of the rarity of this affliction, autosomal dominant inheritance is strongly suggested. The individual marked by an asterisk is only six years old.

Summary

Hereditary general gingival hyperplasia has been described as an overgrowth of gingival tissue covering much or all of the teeth. In one family line this condition is traced through three and possibly four generations, affecting both sexes, with a ratio of affected to unaffected individuals near 1:1. From this evidence it is reasonable to assume that the anomaly is inherited as a simple autosomal dominant.

Literature Cited

1. Buchner, H. J. *Jour. Am. Dent. Assn.* 24:2003-2007. 1937.
2. Prinz, H., and S. S. Greenbaum. Diseases of the Mouth and Their Treatment. Lea and Febiger, Philadelphia. Pp. 162-167. 1939.
3. Ruggles, S. D. *Jour. Am. Med. Assn.* 84: 20-24. 1925.
4. Stones, H. H. Oral and Dental Diseases. E. and S. Livingstone, Edinburgh. Pp. 587-589. 1948.
5. Thoma, K. Oral Pathology. C. V. Mosby, St. Louis. Pp. 1066. 1941.
6. Weski, H. *Deutsche Monatsschrift für Zahnheilk.* 38:557-584. 1920.

◆

American Association of Blood Banks

THE Third Annual Meeting of the American Association of Blood Banks is being planned for October 12, 13, 14 at The Stevens Hotel in Chicago. The program which is being planned will be one which will attract blood bank personnel, hospital executives, pathologists, clinicians, surgeons, and other people interested in the procurement, preservation, and administration of blood and blood derivatives. For further information write the Office of the Secretary, 3301 Junius Street, Dallas 1, Texas.

THE INHERITANCE OF FLECKED AND MARK

In *Nemesia strumosa*

HERBERT PARKES RILEY
University of Kentucky, Lexington, Kentucky

THE inheritance of variegated flowers in which dots, dashes, blotches, or sectors of one color or of white are superimposed upon a colorless background or upon a background of another color has interested botanists for a long time. Several recent discussions of the subject make unnecessary a detailed report on the literature, but a few of the explanations will be reviewed.

Wedge-shaped sectors have often been regarded as the result of an irregular somatic mitosis in a heterozygote by which the two daughter chromosomes bearing the dominant allele go together into the same daughter cell leaving the other daughter cell without the dominant allele and with $2n$-1 chromosomes (Lawrence[5]). All the cells that arise from the deficient cell show the recessive character and cells that arise from the daughter cell with the extra chromosome look like the rest of the flower unless the dominant allele has a cumulative effect. The recessive sectors are small if the aberration occurs late in the development of the flower but are large if the flower is very young when the aberration occurs.

Such sectors have been found by the writer in flowers of *Nemesia strumosa*.[8,9,11] They include white sectors in orange-colored flowers, yellow sectors in white flowers, orange sectors in red flowers, white sectors in purple flowers, a purple sector bounded by narrow white sectors in a red flower, and a blue sector in an orange flower.

Variegated types with many small dots or flecks have also been reported. Gregory[3] reported that such a flecked pattern appeared to be recessive to self-color in *Primula sinensis*. Punnett[6] has reported a series of three alleles in the sweet pea. Gene G is the top dominant and conditions self-color. Allele G' is an allele for flaking and g for white: but G' is not completely dominant over g. Lawrence[5], in addition to sectorial chimaeras, has also found a type of mosaicism in *Dahlia variabilis* in which flecks and streaks and sometimes larger areas of anthocyanin are found. He has reported that the inheritance of this type of mosaicism appears to be Mendelian and is not to be attributed to somatic segregation.

In *Delphinium ajaxis* Demerec[2] has found variegated flowers which appear to result from a gene which mutates frequently in somatic tissue from a recessive to a dominant condition, resulting in many specks and larger patches of purple on otherwise rose-colored flowers. Largely from this work has arisen the concept of mutable genes according to which some genes are thought to have a relatively high mutation rate, mutating frequently to an allele either in germinal or somatic tissue or frequently in both.

The concept of mutable genes has been used to explain flecking in flowers of *Portulaca grandiflora*. Blakeslee and Warmke[1] have suggested that red flecks are the result of somatic mutations from the recessive to the dominant. Imai and Kanna[4] have reported a more complicated situation. A certain gene when homozygous produces orange self-colored flowers; an allele when homozygous produces cream-colored flowers; the heterozygote has yellow flowers. The cream gene mutates frequently to the orange allele in both somatic and germinal tissue. For this reason the heterozygote has yellow flowers with orange-colored stripes, where somatic mutation has produced the homozygous orange color. For the same reason flowers of plants homozygous for cream are cream-

colored but with yellow stripes, when the tissue is heterozygous due to somatic mutation. In the yellow regions of these flowers small orange stripes appear, which represent later mutations of the second allele. Mutations of a similar nature occur also in germinal tissue.

Materials and Methods

As mentioned elsewhere[8], the writer's studies on *Nemesia strumosa* have been based on seeds obtained from commercial seed houses upon several different occasions, but it was only in the original packet of mixed seeds that either the flecked or the mark types were found. All the subsequent sowings failed to include either of these types and, unfortunately, the original lines died out before several desired tests could be made.

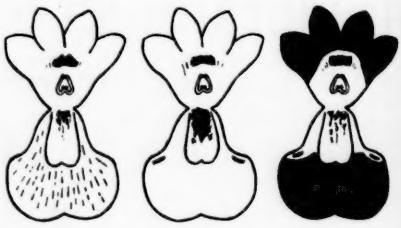
Observations on Flecked

The flecked type appeared first in the offspring of several crosses among the original plants. It was first seen among the orange-flowered lines but was found the following year in plants with the white background.

In the orange-colored plants the flecked character appears as numerous very short streaks or dots of red on the inner side of the lower lip of the corolla (Figure 7*A*), but in white-flowered plants the color is purple and seems to be always in dots rather than in dashes (Riley[7]). The number and arrangement of the flecks vary considerably in different flecked plants and even on flowers of the same plant.

Preliminary studies seemed to indicate that flecked was recessive to non-flecked but in some families from crosses between two non-flecked plants or between a flecked and a non-flecked there was a significant deficiency of the flecked type. Of twenty segregating families from two non-flecked parents six deviated significantly from a 3:1 ratio and in several others the ratio of the deviation to the standard error was close to 2.00. Of seven backcrosses, two differed significantly. A further examination of the first six deviating families showed that one segregated into an almost perfect 7:1 ratio and the other five were much closer to 15:1 than to 3:1. Also, the two backcross families which deviated significantly were closer to 3:1 than to 1:1 ratios. These five families suggest the possibility of duplicate factors.

Assuming that duplicate factors might be operating with the double recessive determining the flecked type, genotypes were assigned which would account for the deviating families and the various lines were traced out consistently. On this basis, three families from non-flecked parents should segregate 3:1, eight should segregate 7:1, and nine should segregate into 15:1 ratios. These families are listed in Table I. The first three are the same under



FLECKED AND MARKED PATTERNS

Figure 7

A—flecked. *B*—mark on an orange (*O c*) background. *C*—mark on a red (*O C*) background. The "mark" color is always either red or blue, even when the ground color is orange.

either theory. Of the next eight, seven are in much closer agreement with the theory of duplicate factors even when they do not deviate significantly from a simple monohybrid ratio. Eight of the next nine families are much closer to 15:1 than to 3:1 ratios. Four of the backcross families should segregate 1:1 on either theory. The other three should fall into a 3:1 ratio if duplicate genes are operating, and all three families are much closer to 3:1 than to 1:1 ratios.

All the families tested agree with the theory of duplicate genes. As Shull[12] pointed out many years ago, a good test for duplicate genes is an analysis of selfed F_2 plants. This critical test could not be used to test the flecked condition because *Nemesia strumosa* is self-sterile and an F_3 cannot therefore be produced by selfing F_2 plants.

There is no evidence for linkage with the self-sterility alleles. Three of the families in which there is no self-sterility allele common to the two parents segregate into good 7:1 ratios and the families in which gametic elimination does occur do not follow consistently any scheme of linkage. Unfortunately data are insufficient to test for linkage with the other genes reported elsewhere.

As has previously been mentioned[10] the relation between the flecked condition and the eyebrow gene is the same as that between *C* and *E*. The gene for anthocyanin pigmentation, *C*, is epistatic to eyebrow, for all plants with *C* show the eyebrow character even when genotypically *cc*. Similarly, all the flecked plants that have been found are phenotypically eyebrow even in families in which the non-flecked plants show segregation for eyebrow. Nine families had no *C* gene and segregated for flecked and for eyebrow. In these families (Table II) there were 57 flecked plants and all were phenotypically eyebrow although the non-flecked plants were segregating 1:1 or 3:1 for eyebrow and non-eyebrow. In seven families segregating for *C* there were 32

flecked plants and all were phenotypically eyebrow. Apparently, then, the double recessive flecked type is epistatic to the non-eyebrow gene. The regular segregation into non-flecked and flecked in the *cc* plants of these families and the failure to identify *C* flecked phenotypes indicates that *C* is epistatic to flecked.

Observations on Mark

The "mark" character has appeared in several families of the writer's original strains but only in families with the orange background. In plants lacking the general anthocyanin pigmentation, the mark type consists in a short line or dash (Figure 7*B*) or series of dots on each side of the lower lips near the tube. The color is either red or blue even on the orange background and sometimes fades from red to bluish-gray as the flower ages. In plants with the *C* gene the mark appears as two lines or series of dots (Figure 7*C*) and each mark is always surrounded by a small colorless area. Apparently the mark type includes not only the dashes of color but also the suppression in *C* plants of anthocyanin in cells surrounding the mark. The effect of the mark type in *oo* plants is not known.

The infrequency of mark types in segregating families suggested early that mark was recessive. The seven families in which it appears all trace back to one family, 3001, in which there were no plants of the mark type. Six of the families resulted from crosses between two non-mark plants while one was a backcross. However, when the observed ratios are compared with the expected 3:1 or 1:1 ratios each deviates significantly. Of the six families from two non-mark parents, one agrees well with a 7:1 ratio and four do not deviate significantly from a 15:1 ratio. The family from a cross between a mark and a non-mark deviates significantly from a 1:1 ratio but is very close to a 3:1 ratio (Table III). Further, on the basis of a digenic system all six families are consistent. The seventh is expected to segregate into a 3:1 ratio on either theory. The close agreement of six families with the expectation based on duplicate factors and the fact that the seventh does not agree with either theory afford strong evidence that the mark type results from the interaction of two duplicate recessive genes.

TABLE I. Observed ratios of non-flecked *(F)* to flecked *(f)* with the expected ratios on the basis of (a) a single recessive gene and (b) duplicate genes. The grouping is by genotypes according to the theory of duplicate genes.

Family	Parents	Observed		Expected if monogenic			Expected if duplicate genes		
		F	*f*	*F*	*f*	dev/S.E.	*F*	*f*	dev/S.E.
				$F_1f_1\ f_2f_2 \times F_1f_1\ f_2f_2$ (3:1)					
3002	2901(1) × 2901(11)	43	12	41.25	13.75	0.55	41.25	13.75	0.55
3221	3112(3) × 3112(6)	9	1	7.50	2.50	1.09	7.50	2.50	1.09
3222	3112(6) × 3112(3)	30	11	30.75	10.25	0.27	30.75	10.25	0.27
Total		82	24	79.50	26.50	0.56	79.50	26.50	0.56
				$F_1f_1\ F_2f_2 \times F_1f_1\ f_2f_2$ (7:1)					
3001	2901(1) × 2901(7)	16	1	12.75	4.25	1.81	14.88	2.12	0.83
3003	2901(1) × 2901(15)	76	12	66.00	22.00	2.46	77.00	11.00	0.32
3015	2901(14) × 2901(1)	15	1	12.00	4.00	1.73	14.00	2.00	0.76
3103	3001(6) × 3001(22)	38	6	33.00	11.00	1.74	38.50	5.50	0.23
3225	3103(1) × 3103(5)	14	2	12.00	4.00	1.16	14.00	2.00	0.00
3226	3103(5) × 3103(3)	20	2	16.50	5.50	1.72	19.25	2.75	0.48
3375	3215(15) × 3215(7)	23	5	21.00	7.00	0.87	24.50	3.50	0.86
3376	3215(15) × 3221(2)	21	4	18.75	6.25	1.04	21.88	3.12	0.53
Total		223	33	192.00	64.00	4.47	224.00	32.00	0.19
				$F_1f_1\ F_2f_2 \times F_1f_1\ F_2f_2$ (15:1)					
3109	3012(1) × 3012(4)	89	4	69.75	23.25	4.61	87.19	5.81	0.78
3110	3012(4) × 3012(1)	14	2	12.00	4.00	1.16	15.00	1.00	1.03
3215	3111(5) × 3112(10)	21	1	16.50	5.50	2.22	20.62	1.38	0.33
3217	3111(3) × 3111(4)	11	1	9.00	3.00	1.33	11.25	0.75	0.30
3206	3112(10) × 3111(4)	3	0	2.25	0.75	1.00	2.81	0.19	0.45
3211	3111(4) × 3111(5)	16	0	12.00	4.00	2.31	15.00	1.00	1.03
3212	3111(4) × 3112(10)	12	0	9.00	3.00	2.00	11.25	0.75	0.89
3214	3111(5) × 3111(4)	7	0	5.25	1.75	1.54	6.56	0.44	0.69
3218	3111(4) × 3111(3)	16	0	12.00	4.00	2.31	15.00	1.00	1.03
Total		189	8	147.75	49.25	6.78	184.67	12.31	1.26
				$F_1f_1\ f_2f_2 \times f_1f_1\ f_2f_2$ (1:1)					
3105	3001(8) × 3001(22)	10	10	10.00	10.00	0.00	10.00	10.00	0.00
3106	3001(22) × 3001(8)	6	3	4.50	4.50	1.00	4.50	4.50	1.00
3321	3103(52) × 3112(9)	2	4	3.00	3.00	0.82	3.00	3.00	0.82
3322	3112(9) × 3103(52)	26	17	21.50	21.50	1.37	21.50	21.50	1.37
Total		44	34	39.00	39.00	1.13	39.00	39.00	1.13
				$F_1f_1\ F_2f_2 \times f_1f_1\ f_2f_2$ (3:1)					
3223	3103(3) × 3103(52)	17	8	12.50	12.50	1.80	18.75	6.25	0.81
3224	3103(52) × 3103(1)	22	6	14.00	14.00	3.02	21.00	7.00	0.44
3362	3103(52) × 3103(40)	35	4	19.50	19.50	4.97	29.25	9.75	2.13
Total		74	18	46.00	46.00	5.83	69.00	23.00	1.20

Discussion

Whenever self-sterile plants are studied, the possibility must be considered that any modified ratios are the result of linkage with self-sterility alleles and consequent gametic elimination. If the modified ratios are found among families in which there is no common self-sterility allele, there is a high probability that there is another cause but if the disturbed ratios are found only in families whose parents had an allele in common and if families segregating into four self-sterility classes do not show disturbed ratios, the presumption is in the other direction. The flecked type shows 7:1 ratios in three families from parents with no common allele and therefore is probably not the result of a single recessive gene linked with the self-sterility alleles. Unlike flecked, however, the mark gene did not appear in any of the families the parents of which had no common self-sterility allele.

If the disturbed ratios are the result of a single recessive gene which is linked with the self-sterility alleles, the data from all the families taken together indicate that there is 13.6 percent crossing over. If two heterozygotes are crossed which have a common self-sterility allele and if *m* is linked with the common allele in the male, the offspring should be in the ratio of 0.932 *M* : 0.068 *m*. If there are duplicate genes and no linkage with the *s* genes, the offspring from two plants heterozygous for each gene should be in the ratio of 0.9375 *M* : 0.0625 *m*. These two ratios are so alike that it would be virtually impossible to decide between the two theories from these crosses. Families 3103, 3105, 3106, and 3362 agree well with either ratio. If the *M* is coupled with the common *s* allele in the male and both parents are heterozygotes, the ratio of the offspring should be 0.568 *M* : 0.432 *m*. Family 3226 agrees well with this ratio. On the basis of duplicate factors the origin of this family indicates that it should segregate 3:1, but it differs significantly from this explanation. If the female is mark and the male heterozygous with the *m*

TABLE II. Segregation for (*E*) eyebrow and non-eyebrow (*e*) in families also segregating for flecked (*f*); some are segregating for colored (*C*) as well. Note that all colored and flecked plants are phenotypically eyebrow.

Genotypes of parents			No. families	Offspring			
				CE	*cfE*	*cFE*	*cFe*
cc × *cc*; *OO* × *oo*; *Ee* × *ee*;	segregating	3*F*:1*f*	3	0	21	55	45
cc × *cc*; *OO* × *oo*; *Ee* × *ee*;	"	7*F*:1*f*	1	0	1	6	9
cc × *cc*; *Oo* × *oo*; *Ee* × *ee*;	"	1*F*:1*f*	1	0	17	10	16
cc × *cc*; *oo* × *oo*; *Ee* × *Ee*;	"	3*F*:1*f*	2	0	12	29	10
cc × *cc*; *oo* × *oo*; *Ee* × *Ee*;	"	15*F*:1*f*	2	0	6	81	22
Cc × *cc*; *OO* × *O*–; *Ee* × *ee*;	"	1*F*:1*f*	2	45	13	2	13
Cc × *cc*; *OO* × *O*–; *Ee* × *ee*;	"	7*F*:1*f*	3	60	9	33	35
Cc × *cc*; *OO* × *O*–; *Ee* × *Ee*;	"	3*F*:1*f*	1	15	8	17	0
Cc × *Cc*; *OO* × *O*–; *Ee* × *Ee*;	"	7*F*:1*f*	1	44	2	20	0

TABLE III. Observed ratios of non-mark (*M*) to mark (*m*) with the expected ratios on the basis of (a) a single recessive gene and (b) duplicate genes. The grouping is by genotypes according to the theory of duplicate genes.

Family	Parents	Observed *M*	Observed *m*	Expected if monogenic *M*	Expected if monogenic *m*	Expected if monogenic dev/S.E.	Expected if duplicate genes *M*	Expected if duplicate genes *m*	Expected if duplicate genes dev/S.E.
		M_1m_1 M_2m_2 × M_1m_1 M_2m_2 (15:1)							
3103	3001(6) × 3001(22)	76	3	59.25	19.75	4.36	74.06	4.94	0.90
3105	3001(8) × 3001(22)	43	1	33.00	11.00	3.45	41.25	2.75	1.09
3106	3001(22) × 3001(8)	30.	0	22.50	7.50	3.16	28.12	1.88	1.41
3362	3103(52) × 3103(40)	38	1	29.25	9.75	3.24	36.56	2.44	1.24
		m_1m_1 M_2m_2 × M_1m_1 M_2m_2 (7:1)							
3223	3103(3) × 3103(52)	36	4	30.00	10.00	2.19	35.00	5.00	0.48
		M_1m_1 m_2m_2 × m_1m_1 M_2m_2 (3:1)							
3226	3103(5) × 3103(3)	40	26	49.50	16.50	2.70	49.50	16.50	2.70
		m_1m_1 m_2m_2 × M_1m_1 M_2m_2 (3:1 backcross)							
3373	3226(7) × 3226(1)	25	8	16.50	16.50	2.96	24.75	8.25	0.18

gene linked with the common allele, the ratio of the offspring should be 0.864 *M* : 0.136 *m*. If duplicate factors are operative and if the non-mark type is a double heterozygote, the offspring should segregate 3:1. The only family from a mark female and non-mark male, family 3373, fits a 3:1 ratio almost exactly and fits the other ratio much less well. The evidence taken as a whole is not conclusive for either theory. However, if there is a single gene linked with the *s* alleles, it must be assumed that of four plants of family 3103 which have been used to produce later generations, one chromosome of two plants and the two chromosomes of a third are cross-over chromosomes and that of two plants of family 3226, one has a cross-over chromosome. This is a much higher percentage of plants with cross-over chromosomes than would be expected from the percentage of crossing over. The theory of duplicate genes, then will be adopted as a tentative hypothesis.

If mark is linked with the self-sterility alleles, it might show linkage with the gene for buff. The only families segregating for both mark and buff are 3103, 3105, 3106, and 3362. These agree equally well with the theory of duplicate genes not linked with *s* or *bu* but also with that of a single gene on the same chromosome with the genes in the order *m-s-bu*.

Summary

The flecked type of flower in *Nemesia strumosa* has numerous short dashes and dots of red or purple on the inner side of the lower lip of the corolla. This character appears to be the result of duplicate factors, the flecked type being the double recessive. Flecked plants always have the large spot of anthocyanin on the upper lip above the stamens and the numerous deep purple spots on the lower part of the tube which are characteristic of the eyebrow gene even when genotypically *ee*.

The mark type has a short line of color on each side of the lower lips near the tube. In *C* plants this line of color is surrounded by a colorless space that separates it from the rest of the color on the lower lips. This character also appears to result from duplicate genes although several other explanations have not been definitely ruled out.

Literature Cited

1. Blakeslee, A. F. and H. E. Warmke. *Science* 90:411-412. 1939.
2. Demerec, M. *Jour. Genet.* 24:179-193. 1931.
3. Gregory, R. P. *Jour. Genet.* 1:73-132. 1911.
4. Imai, Y. and B. Kanna. *Genetica* 17: 27-31. 1935.
5. Lawrence, W. J. C. *Jour. Genet.* 24:257-306. 1931.
6. Punnett, R. C. *Jour. Genet.* 32:171-177. 1936.
7. Riley, H. P. *Genetics* 25:133 (abstract). 1940.
8. ———— *Bull. Torrey Bot. Club* 72: 435-453. 1945.
9. ———— *Bot. Gaz.* 107:32-52. 1945.
10. ———— *Jour. Hered.* 39:170-172. 1948.
11. ———— *Bot. Gaz.* 111:43-52. 1949.
12. Shull, G. H. *Zeit. ind. Abst. Vererb.* 12:97-149. 1914.

THE TREND OF SCOTTISH INTELLIGENCE

THIS little book* is the outcome of the sincere efforts of the Scottish Educational Research Council in attempting to get at the truth of a question so frequently asked today—"Is the intelligence of the population falling?" At the same time, the Council is hoping to obtain sufficient additional information, mostly sociological, in the hope of gaining some insight into the causal agencies of any intelligence trend that might be established.

Actually, this volume is not a detailed report on the results of a population study, but presents the problems at hand in administering such a study. In a somewhat chronological, and uninspiring manner, the historical background, organization, administration, and actual procedure are presented. Nevertheless,

*The Scottish Council for Research in Education v. + 151 pp. University of London Press Ltd. London, 1949.

this is of particular value, since it affords the reader an opportunity to grasp the methodology necessary to this or other studies of this nature. The statistical reliability of the results is also evident from these discussions. The new material is derived from several intelligence tests and their analysis with respect to family size and previous surveys made.

In 1932, a broad survey of eleven-year old school children was made by group tests. One thousand of these children were given individual tests. In June of 1947, the present survey was made on children of the same age, with about 1200 being tested individually. The individual tests were more to confirm the adequacy of the group tests which included 87,000 and 71,000 children in 1932 and 1947, respectively. Additional information on 10 percent of the group was obtained with respect to socio-economic status, medical history, and so forth. The results of this latter investigation are not presented. The two most outstanding features from the intelligence analysis were that the intelligence of children decreases with increasing family size, and that no significant change in Binet I.Q. has occurred in the fifteen year period, 1932-47.

A third, rather fascinating feature that was brought to light was the tendency, in families of three, four, and sometimes five children, for the first and last offspring to be slightly more intelligent. Sampling difficulties make this last point less reliable, but the former two findings appear to be extremely well established. It is intriguing, too, to conjecture on the causes for this negative association of family size and intelligence; the authors, as well, like children on Christmas Eve, seem hardly able to restrain themselves. But they remain non-committal until they have analysed the sociological data, which will be reported in a subsequent volume. The genetic and environmental effects that may be brought out should prove most fascinating.

In spite of the many phases of the problem, and the almost unsurmountable administrational difficulties, the approach to this internationally significant question is an excellent start. It is the Council's sincere wish that this type of survey be continued for many years and that each additional survey profits from the shortcomings of the last. Undoubtedly, many criticisms to the particulars of the procedure could be made, but, in the words of Dr. Thomson, Chairman of the Survey Committee—"We must not make the better the enemy of the good."

D. GRAHN

Iowa State College

✦

Research Facilities for Guest Investigators

THE Jackson Laboratory offers each summer coöperative research facilities to a limited number of mature investigators. This offer is particularly addressed to younger workers with interests related to the Laboratory's research programs in growth and social behavior of animals, and whose research cannot be satisfactorily carried out elsewhere.

Summer investigators are selected on the basis of suitability of the research plan submitted and on the capability of the author for independent research. In general this implies training of the Ph.D. grade or equivalent experience, but graduate students working on thesis problems may apply. Because of the problems inherent in working with mammalian genetic material, each visiting investigator is sponsored by a resident staff member who assists in coöperative planinng for the summer's work. Small grants covering extra expenses are sometimes awarded.

Application blanks may be obtained by addressing the "Summer Investigator's Committee, Box 847, Bar Harbor, Maine." Action will be taken on completed applications as received. The period of residence for 1950 will extend approximately from June 12 to September 3rd.

SELF-INCOMPATIBILITY STUDIES IN GUAYULE

I. Pollen-Tube Behavior

D. U. GERSTEL AND MARY E. RINER*

DURING the early forties it was discovered that the reproductive processes of guayule (*Parthenium argentatum* Gray) are very complex. More recently, Bergner[4], and Stebbins and Kodani[21] found that the species consists of a polyploid series with a lowest somatic chromosome number of 36. Wild diploids reproduce sexually and are self-incompatible[11,18], while the wild polyploids are pseudogamic and facultative apomicts; in these their own as well as unrelated pollen can induce the seed to develop[11,18].

This combination of sexuality in the diploids with apomixis in the polyploid representatives of a genus is quite common (for an extensive list see Gustafsson[12]); but the genetic mechanism controlling the relations between self-incompatibility, polyploidy, apomixis and their opposites are not well understood in any one case.

The present paper deals with one of these factors, namely self-incompatibility, and is concerned only with its histological aspect without giving the results of inheritance studies which are under way.

Materials and Methods

The diploid plants of guayule used in this investigation were derived from the collections of Dr. W. B. McCallum and named SP-7, SP-8, SP-11 and SP-12. The locality of their wild origin could not be ascertained. They had 36 chromosomes with the exception of SP-12 which had two additional microchromosomes (counts by Dr. D. G. Catcheside).

Artificial tetraploids (47-76-8;-10;-27;-43;-63) were obtained by treating germinating seeds with .03 percent aqueous colchicine solution for three hours at 70° F. The chromosomes of the PMC's were counted to confirm the polyploid nature of the plants ($2n = 72$) and observation of the size of the pollen grains on the preparations used in the experiment served as an additional check.

Strain 593 is a commercial apomictic variety of Dr. McCallum's selection and has 72 chromosomes. The four plants used could not be distinguished morphologically. The *Parthenium* inflorescence has been described in detail by Artschwager[1] and requires no elaboration here. It consists of several flower heads which in turn are composed of five stigma-bearing ray florets and many anther-bearing disk-florets.

Several techniques were tried before one suitable for the study of pollen-tube formation in this genus could be found. The main difficulty consisted in preventing the loss of pollen grains from the stigma and it was therefore necessary to reduce the number of manipulations to a minimum.

The procedure finally chosen consisted of the following steps: The inflorescences to be used in pollinations as male and as female parents were bagged at the same time under porous paper bags. Approximately five days later the heads were cut off and brought into the laboratory, at which time the older heads showed the desired stage of maturity. Except where noted below, stigmata with lobes which had just spread were used for the tests. As *Parthenium* is slightly protogynous, few pollen grains had fallen by this time from the anthers of a head upon its own stigmata, but special precautions to protect stigmata from their own pollen were usually considered unnecessary. Cases where controlled emasculation was practiced will be mentioned below. The florets of the male parent were handled with forceps and their pollen dusted on all five stigmata of one head to be used as female. The pedicel bearing the treated head was planted in moist sand in an unstoppered vial for an hour at roughly 75° F. The stigmata with styles (Figure 7*A*) were then removed by forceps and transferred to the stain-fixative.

The stain-fixative consisted of equal parts of water, glycerine, phenol and lactic acid ("lactophenol") to which traces of acid fuchsin and light green had been added to impart the mixture a full purplish color. The tissues were left in this mixture for about an hour, heated gently and then transferred to clear lactophenol and covered.

After 24 hours the tubes in the stigma had stained a full red and stood out in fair con-

*Associate Geneticist and, formerly, Agent (Jr. Cytologist), respectively, Natural Rubber Research Station, Bureau of Plant Industry, Soils and Agricultural Engineering, Agricultural Research Administration, U. S. Department of Agriculture, Salinas, California.

trast against the surrounding tissue. In the lower style, however, the tubes could not be recognized after this treatment. Slides prepared in this fashion lasted many months. The technique was adapted from the procedures employed by Atwood[2] and Buchholz[6].

Compatible Matings

The behavior of compatible pollen tubes is pictured in Figures 8*A* and *C*. The tubes show a sharp bend where they enter the conducting tissue. There they become aligned parallel to the axis and usually grow downward (occasionally a compatible tube will grow in the upward direction). This sharp bend is characteristic for the compatible tubes and has not been observed among the incompatible ones which are occasionally lodged between the papillae of the stigmatic epidermis. The approximate location of the conductive tissue (*pt*) to which the tubes are restricted can be observed in Figure 8*A*.

The tubes are by no means of uniform appearance. After heavy pollination many of them end within the stigma. They either taper off, produce a bulb, or burst. Only a fraction reaches the lower style. There is also great variation in the thickness of the tubes; some appear almost threadlike under 350× magnification while others are about as wide as the cells of the conducting tissue. Tubes may grow to considerable length without the grain becoming emptied.

In any pollination work it is necessary to determine the period during which the stigma is receptive. In the following the critical time limits are described.

As in other Compositae[13] the receptive part of the stigma of *Parthenium* is restricted to the upper surface of the stigmatic lobes. These are folded together tightly until some time after they have emerged from the corolla and the receptive area remains covered until the lobes separate. Consequently, efforts to pollinate before separation of the lobes failed and no tubes could be observed in any instance, even though the outer surfaces of the stigmata were thoroughly covered (Table I, a).

That this is due to the condition mentioned and not to immaturity of the stigmata is shown by columns b) and c) in the table. If the lobes were pried apart or one of the branches was cut off with a razor blade and pollen from a mate then brushed onto the exposed inner papillae, germination ensued and tubes could subsequently be observed. All the data in Table I were obtained about one day before the florets would normally have opened.

Therefore, stigmata are receptive even before the florets open, but without proper manipulation pollination will remain ineffective until the lobes have separated. The results of selfings, after the same treatments, are also given in the table and will be discussed below.

To determine the time when stigmata cease to be receptive, open heads were cross-pollinated at three different stages of development: (1) just after the stigma lobes had separated, which stage slightly precedes the dehiscence of pollen from the outermost whorl of male florets on the same head; (2) intermediate stage, coinciding more or less with the dehiscence of pollen from the inner whorls of male flowers; and (3) after all male flowers had shed their pollen, but before any sign of necrosis appeared on the stigma. The time elapsed between (1) and (3) is roughly three days under greenhouse conditions. The results of the experiment are summarized in Table II.

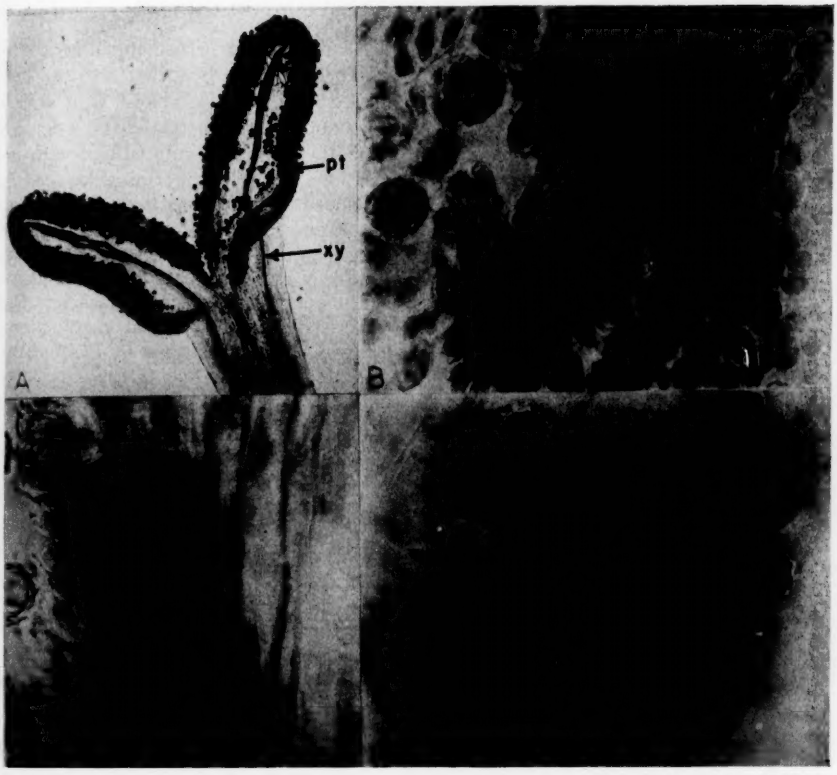
Obviously, the exact amount of pollen applied to each stigma could not be measured; but the pollinations were equalized by using excess pollen for each stigma until the receptive area was completely covered. The data indicate a decline at the third stage after all pollen is shed, but some receptivity apparently remains until necrosis sets in. As only one tube needs to reach the single ovule, pollination may result in fertilization as long as the stigma remains white and turgid.

Incompatible Matings

The left side of Table III shows that incompatible pollen can germinate on the stigma (Figure 8*D*). Counts of germinated and ungerminated grains were made on stigmata which were completely covered with pollen, either their own (50 cases) or foreign pollen (58 cases). Two plants, SP-11 and SP-12, were used; selfings and reciprocal matings between them were made and germinating pollen grains counted. The experiment was performed during 1947 and 1948; in the latter year emasculation was practiced but not in the earlier one. The incompatible pollinations showed a considerably lower germination of pollen grains than the compatible ones—9.4 percent as against 65.0 percent on the average.

After germination the next stage in the life cycle of a male gametophyte is the entrance of the tube into a stigma. In guayule it was found that incompatible tubes fail to penetrate beyond the papillae of the stigma and do not enter its inner tissues. These incompatible tubes reach a length up to four pollen diameters, but usually they are shorter. They may either end in a bulb or taper to a point (Figure 8*D*). On very rare occasions one tube was found inside the stigma in incompatible matings; such an event could be due either to contamination from the air during hand pollination or mutation from an incompatible to a compatible allele[15].

Data to demonstrate the absence of tubes from selfings are not assembled here in a special table, but can be found in the lower part of Table I, where a total of 76 stigmata were pollinated with pollen from the same heads and showed not a single tube, while 131 cross-matings showed tubes in 117 cases. Similarly, in Table V, fourth and fifth row, 51 matings between SP-7 and SP-8 showed tubes in 45 stigmata while



Photographs by Wm. Mishanec

POLLEN-TUBE GROWTH

Figure 8

A—Entire stigma with compatible pollen and tubes. ×58. (xy = xylem, pt = pollen tubes). *B*—Incompatible pollination. ×900. *C*—Section of stigma with compatible pollen and tubes. Turn-back tube on lower right. × 900. *D*—Incompatible pollination with abortive tubes. ×900.

146 selfings of their progeny plants produced not a single tube. It may be noted that the data in Table I were derived from bud pollinations while those in Table V resulted from open florets. Finally, during the summer of 1948, pollinations on approximately 5,000 stigmata of progenies between SP-7 and SP-8 were performed for a genetical analysis of incompatibility in that group. Here again, selfings resulted in the failure of pollen tube growth (with the exceedingly rare exceptions noted above). Also certain cross-matings consistently failed to produce tubes, while they were formed in others just as consistently. While the presentation of these data and their analysis will be reserved for the future, it should be mentioned here that selfings and incompatible cross-matings behave alike in failing to pass the threshold of the stigma surface.

Pseudo-fertility

Attempts were made to induce self-fertilization in our material by methods which had successfully produced "pseudo-fertility" in other genera. Thus, in *Nicotiana*[7], cabbage[17], and many other plants selfings of unopened buds had resulted in seed set. The data in Table I, which were obtained from closed florets about one day before their opening, two days before the natural separation of the lobes and as early as they could be handled, indicate the failure of this technique in guayule. The stigmata of 76 buds of SP-11 and SP-12 were selfed, after their receptive areas had been properly exposed to the pollen, but none showed any tubes at the time of examination. The 131 control cross-matings in the first two rows of the table between SP-11 and SP-12 produced tubes in 117 cases. Thus, there is

apparently no bud-fertility in guayule.

Sears[19] employed a different technique to overcome the effects of self-incompatibility in broccoli, where an inhibitory barrier against selfing occurs on the surface of the stigma, just as in guayule. He found that removal of a layer of stigmatic surface, or even of the entire stigma plus considerable portions of the style permitted incompatible pollen grains to germinate on the stump. However, in guayule no germination even of compatible grains takes place on the cut surfaces of the style, as indicated in Table IV and therefore no functioning of incompatible grains can be expected.

The methods used in our attempts to induce selfings are admittedly crude and it is not impossible that their refinement will lead to a better success.

Seasonal pseudo-fertility as reported for *Nicotiana*[8], *Taraxacum kok-saghyz*[27] and others apparently does not prevail in guayule. Matings for the genetical analysis were started, during two seasons, as soon as the first flowers appeared in the cultures in early June and continued to the very end of the flowering season in mid-October, both outdoors and in the greenhouse. Incompatible and compatible reactions which followed the same pattern were obtained during the entire season.

Polyploids

As mentioned in the introduction, it had been shown by other authors that the triploid and tetraploid wild collections and the commercial strains derived from them are self-compatible, with indications that less germinable seed is produced after selfing than after crossing even here[18]. The pollen-tube test also demonstrates this self-compatibility, (Table V); 31 stigmata from four plants of the commercial tetraploid variety 593 were selfed by the usual method and tubes could be discerned in 12 of them. Also tested were five tetraploid plants obtained after colchicine treatment from the cross SP-8 × SP-7. None of their 28 selfed stigmata showed any tubes (row 1 in Table V). That this failure is not due to low fertility of the pollen is shown by the second row in the table, where 20 compatible matings of four of

TABLE I.—Pollination of unopened florets

Parents	a) Stimata not manipulated		b) Stigma lobes forced apart		c) One lobe cut off*	
	No. pollinated	Stigmata with tubes	No. pollinated	Stigmata with tubes	No. pollinated	Stigmata with tubes
SP-11 × SP-12	20	0	45	42	27	21
SP-12 × SP-11	20	0	37	36	22	18
SP-11 selfed	——	——	19	0	17	0
SP-12 selfed	——	——	20	0	20	0

*Tubes grew only on remaining lobes, not on cut surfaces.

TABLE II.—Time of receptivity of stigmata (SP-11 × SP-12)

Stage of Maturity	Mean* number of pollen tubes per stigma and standard error	
	1947	1948
1	45.8 ± 6.27	18.2 ± 2.23
2	31.4 ± 3.42	33.8 ± 4.39
3	5.8 ± 1.63	7.2 ± 1.02

*Each mean represents 20 stigmata.

TABLE IV.—Stigma mutilation

Mating	—Stigma removed— Stumps pollinated	Styles with germinated grains	Stigma retained (controls) Stigma pollinated	Stigmata with germinated grains
SP-11 × SP-12	19	0	10	9
SP-12 × SP-11	20	0	9	9

TABLE V.—Self-incompatibility of artificial tetraploids and their diploid relatives and self-compatibility of a commercial tetraploid variety

Chromosome number		No. of plants	Total stigmata pollinated	Stigmata with tubes
72 selfed	Colchicine doubled, ex SP-8 × SP-7	5	28	0
36 × 72	Parent × progeny above	4	20	19
72 × 36	Progeny above × parent	2	15	14
36 selfed	Sibs of above tetraploids	16	146	0
36 × 36	Parents of above	2	51	45
72 selfed	Commercial var. 593	4*	31	12

*Four morphologically identical apomicts.

TABLE III.—Germination of pollen grains on self- and on cross-pollinated plants

Year	Self-pollinated Plant no.	Total stigmata	Total grains	Grains germinated	% germinated	Year	Cross-pollinated —Plant No.—	Total stigmata	Total grains	Grains germinated	% germinated
1947	SP-11	10	268	29	*10.8*	1947	SP-11 × SP-12	14	810	623	*76.9*
	SP-12	10	297	37	*12.5*		SP-12 × SP-11	14	562	454	*80.8*
1948*	SP-11	15	1432	226	*15.8*	1948*	SP-11 × SP-12	15	551	343	*62.3*
	SP-12	15	2041	86	*4.2*		SP-12 × SP-11	15	1212	619	*51.1*
Totals		50	4038	378	*9.4*	Totals		58	3135	2039	*65.0*

*Emasculation was practised in 1948 but not in 1947.

the 4 *n* plants with the SP-8 parent produced tubes in 19 cases. That the tetraploid stigmata are not at fault either is demonstrated in the third row: 15 compatible stigmata of two of the tetraploid plants were pollinated from their parents (10 of one plant with SP-7 pollen and five of the other with SP-8) and tubes ensued in 14 out of these 15.

The second and third row of Table V represent only a fraction of the crosses made between the *2n* parents and their *4n* progeny and crosses between compatible plants have been deliberately selected for the table. Failure of pollen tubes to grow in the other $2n \times 4n$ and $4n \times 2n$ crosses was not due, however, to any sterility of the floral organs, but to the working of the incompatibility mechanism which affects parent-progeny relations in the tetraploids as well as in the diploids. That these incompatibilities follow a similar pattern in the tetraploids as in the diploids will be shown in the later publication. Here it is only pertinent to demonstrate that the five artificial *4n* plants are self-incompatible and that the results obtained are not confused with any sterility.

Data on selfings of diploid sibs of the tetraploids and matings between their diploid parents are given in rows four and five of Table V for comparison.

Corroborating evidence for the fertility of the *4n* plants came from an observation of their pollen: only a few grains appeared subnormal under the microscope. An actual count of one of them (47-76-8) in aceto-carmine gave 1309 normal (i.e. showing at least one nucleus), 73 subnormal (i.e. empty or shrivelled) and 27 microcytes.

Discussion

Two procedures are suited for a first analysis of the hereditary mechanism controlling self-incompatibility in any species. Of these, counts of seeds set after crossing are adapted where large numbers of seeds are easily produced by a single operation. Examples are *Nicotiana* with its large capsules or Compositae with perfect florets and sizable heads which can be rubbed together in pairs and thereby pollinated in their entirety. In guayule each operation can produce only a single seed and seed setting is relatively low. Hence the pollen tube method is the more practical approach. Also, the results from compatible and incompatible pollinations are very distinct in this plant, whereas in *Nicotiana* tubes are formed from both and only their growth rates are different[7, 9].

Sears[19] has grouped the self-incompatible higher plants in three classes, which are based on the part of the pistil where the male gametophyte is usually inhibited.* These three regions are: 1) the stigma, 2) the style and 3) the ovary. In the latter, fertilization occurs but zygotes fail to develop if the mates are incompatible. Our researches show the guayule belongs to the first class.

We could not demonstrate, as Sears has done in his ingenious broccoli experiment[19] that the reaction in guayule takes place only on the epidermal layers of the stigma. This demonstration was not made, because even compatible tubes failed to grow on mutilated stigmata lacking the epidermis. Perhaps by the method of style-grafts it will be possible to determine whether the stigmatic surface represents the only threshold for incompatible grains.

Another way of classifying incompatibilities is on the basis of their relative strength[12, 19]. In some species, the "weak" types, incompatibility is effective only part of the time, but absent in buds, or during certain seasons (for references see Stout[23]). Here it may also be overcome by shifts in the "interior environment" as by modifiers in *Nicotiana*[5] or *Petunia*[16] and by polyploidy (examples are listed in Gustafsson[12]). On the other hand, a species with such a strong incompatibility reaction as *Oenothera organensis* has not been found to

*While the point has not been completely proven, it is widely assumed that pollen tube failure is due to the formation of an inhibitor, rather than to a lack of stimulation. For a recent attack upon this concept see Straub[26].

show any pseudo-fertility[10] nor is the failure to set seed affected by polyploidy[14]. Of great interest here is the discovery of Lewis[14] that while in selfed 4*n* *Oenothera organensis* generally no seed is set, the inhibition is weakened sufficiently to allow certain tubes to grow partly down the style.

In *Nicotiana,* incompatible and compatible tubes grow at different rates, while in broccoli and guayule the difference between the two is a qualitative one; either they grow down the style or they do not enter the conductive tissue of the stigma at all. This difference in behavior also would seem to be related to the strength of the incompatibility reaction.

Only a few species have been studied from all three aspects. Of these, *Petunia* is weak in every regard, showing pseudo-fertility, self-compatibility of artificial polyploids and a qualitative difference in behavior between compatible and incompatible pollen tubes[24, 25, 28]. *Oenothera organensis* is a strong species on all three counts[10, 14] while broccoli combines a distinctly qualitative reaction with a pronounced pseudo-fertility[19], thus proving that these factors may be independent from each other.

The incompatibility reaction of diploid guayule must be classed among the strongest because no expression of pseudo-fertility could be found, because of the qualitative nature of the reaction and also because artificial polyploidisation had no effect. This statement can be made only with the reservation that weak alleles may be found in the future, inasmuch as in at least one other species (*Nicotiana alata*) East[7] has demonstrated the widely varying strength of incompatibility alleles within the species.

In guayule, the difference between the colchicine induced tetraploids and the, at least partially, self-compatible wild polyploids require further investigation and may have an explanation in the diallelic* nature of the colchicine induced tetraploids. These tetraploids have *S* alleles in the same proportions as the diploid embryos from which they came and might differ in this respect from wild plants. Thus, Atwood[3] obtained in a cross between two diallelic plants of *Trifolium repens* a 3:26 segregation self-incompatible to self-compatible and suggests that this parallels the segregation of diallelic and tri- and tetrallelic plants. The possibility that the self-compatible natural polyploids of guayule are more highly heterozygous for incompatibility alleles than the colchicine induced ones merits study. An alternative possibility is the presence of self-compatibility alleles in the natural material.

The literature of the inheritance of self-incompatibilities abounds with data which did not follow any systematic pattern[8, 20, 22]; the irregularities of the results obtained apparently were due to the weakness of factors easily affected by genetic modifiers or changes in the environment. One may expect that the inflexible incompatibility system of guayule will not present these difficulties and will be more susceptible to genetical analysis.

Summary

1. The work reported here is a necessary preliminary to the study of inheritance of incompatibility in diploid guayule.
2. The stigmata are receptive to pollination even before their lobes unfold but need to be separated artificially for pollen to reach the receptive area. They remain receptive until all male florets of a head have shed their pollen, though receptivity appears to decline towards the end.
3. Incompatible grains germinate at a lower rate than compatible ones and their tubes are unable to penetrate beyond the epidermis.
4. Selfing could not be accomplished by removal of inhibiting stigma tissues because germination of pollen takes place only on the papillae of the receptive area.
5. No seasonal or bud-pseudo-fertility could be observed.
6. Colchicine induced tetraploids are self-incompatible and it is shown that true sterility is not mistaken for incompatibility. Natural polyploids of guayule are self-compatible.

*In Atwood's[3] terminology *diallelic* refers to 4*n* plants with two different *S*-alleles; *triallelics* have three and *tetrallelics* four different *S*-alleles.

7. On the basis of these findings guayule is considered a species with a very strong self-incompatibility mechanism if compared with other self-incompatible flowering plants.

Literature Cited

1. ARTSCHWAGER, E. *U. S. Dept. of Agric. Tech. Bull. 842.* 33 pp. 1943.
2. ATWOOD, S. S. *Amer. Journ. Bot.* 28:551-557. 1941.
3. ———. *Proc. Nat. Acad. Sci.* 30: 69-79. 1944.
4. BERGNER, A. DOROTHY. *U. S. Dept. of Agric. Tech. Bull. 918.* 1946.
5. BRIEGER, F. *Naturwiss.* 15:734-740. 1927.
6. BUCHHOLZ, J. T. *St. Technol.* 6:13-26. 1931.
7. EAST, E. M. *Proc. Nat. Acad. Sci.* 20: 364-368. 1934.
8. ——— and J. B. PARK. *Genetics* 2:505-609. 1917.
9. ———. *Genetics* 3:353-366. 1918.
10. EMERSON, S. *Bot. Gaz.* 101:890-911. 1940.
11. ESAU, KATHERINE. *Hilgardia* 17:61-120. 1946.
12. GUSTAFSSON, Å. Apomixis in Higher Plants. *Lunds Univ. Årsskr. N. F.* Avd. 2. Bd. 43. 370 pp. 1946-1947.
13. KERNER v. MARILAUN. A. Pflanzenleben. II. Die Pflanzengestalt und ihre Wandlungen (Organlehre und Biologie der Fortpflanzung). III. edition by Hansen. XII & 543 pp. Leipzig-Vienna. 1913.
14. LEWIS, D. *Heredity* 1:85-108. 1947.
15. ———. *Heredity* 2:219-236. 1948.
16. MATHER, K. *Jour. Genetics* 45:216-235. 1943.
17. PEARSON, O. H. *Calif. Agric. Expt. Station Bull. 532.* 22 pp. 1932.
18. POWERS, LEROY and ROLLINS, R. C. *Journ. Amer. Soc. Agron.* 37:96-112. 1945.
19. SEARS, E. R. *Genetics* 22:130-181. 1937.
20. SIRKS, M. J. *Mem. Hort. Soc. N. Y.* 3: 325-343. 1927.
21. STEBBINS, G. L., JR. and M. KODANI, *Jour. Hered.* 35:162-172. 1944.
22. STOUT, A. B. *Mem. N. Y. Bot. Gard.* 6:333-454. 1916.
23. ———. *Bot. Rev.* 4:275-369. 1938.
24. ——— and C. CHANDLER. *Science* 96:257-258. 1942.
25. STRAUB, J. *Zeitsch. Naturforsch.* 1:287-291. 1946.
26. ———. *Zeitsch. Naturforsch.* 2b: 432-444. 1948.
27. WARMKE, H. E. *Amer. Nat.* 78:285-288. 1944.
28. YASUDA, A. *Proc. Imp. Acad. Tokyo*„15: 359-361. 1939.

✦

BLOOD GROUP SYMBOLS AND GENES

Some Thoughts on Allelism, Pleiotropy, and Dominance

P. W. WHITING

University of Pennsylvania

IT is customary in genetics to use full-sized letters, higher or lower case according to dominance, to designate a locus on a linkage map or for genes segregating independently. This is convenient when single pairs are concerned but when more than two alleles are found at one locus it is orthodox to employ superscripts. Thus it has come about that the symbol for the locus is more prominent than that for the trait. It would be simpler, as well as easier for the typesetter, to use full-sized letters only, designating the locus by italics, the multiple allelic traits by roman.

These points may be illustrated by the A, B, O blood[1] group genes, the locus for which has been called *L* (Landsteiner) by Strandskov.[2] The symbols *L*A (or *L*A1 and *L*A2 if we wish to distinguish subgroups), *L*B and *L*O thus designate both the locus and the trait, the specific antigen present. For students working problems the italicized letters can be omitted when only one locus is concerned. Strandskov[2] objects to the symbols *A*, A^B and *a* recommended in various texts. These are especially inconvenient for the teacher who, correcting an examination, has to decide from the student's writing whether A^B refers to the gene *L*B or to the blood group AB.

There are a few statements in Strandskov's very clearly presented paper which I believe to be erroneous or inadequate and upon these I would like to comment. Strandskov does not mention the further alternative to antigens D *vs.* d, namely D^u, and to C *vs.* c, namely C^u, C^w, and C^v, in the rhesus series. If Wiener's gene R^z be symbolized as Rh^{DCE} and Wiener's *r* as RH^{dce}, how would these further alternatives be represented? Should the typesetter be provided with super-superscripts? Third, fourth, etc., alternative antigens might be designated by two-letter symbols, a higher and a lower case letter respectively. In order to avoid superscripts altogether a rhesus allele determining the antigens D^w, C^w and E might be written *Rh*DuCwE. Subscripts might also be dispensed with in genetic nomenclature,—for example by symbolizing sucessive generations as P2, P1, F1, F2, etc.

Fisher's "alleles" within alleles I have written in order DCE rather than CDE because

it is supposed that the gene elements have become linearly differentiated in that order. This theory is based, not upon any observed cases of crossing over, but upon gene frequencies in the population and the assumption that the rare types are maintained by single crossing over which is balanced by lethal selection. Double crossovers do not occur within so short a region. One may consider these factors as constituting three series of closely linked genes. However, quoting Strandskov's statement that "a gene is what behaves as a unit in heredity," I would as a matter of convenience call *Rh*DCE, *Rh*DuCwe, *Rh*dCvE, etc., allelic genes with three D alternatives, five C alternatives and two E alternatives, giving 30 possible alleles in the rhesus series.

Thus the "two quite distinct hypotheses for the inheritance of the observed Rh-Hr blood variations" are not as distinct as the heated discussions between Wiener and the Fisher school may seem to suggest. If genic structure is differentiated by reorganization of materials and increased by adjacent repeats of the chromonema it becomes purely a matter of definition whether we are dealing with a single pair of pleiotropic genes or with two genes that are closely linked. If we count 10,000 offspring from a test-cross and find no recombination of the various alternative traits, we say we have a single pair of pleiotropic genes, but if we count 10,001 offspring we may find a crossover and so we would say we have two pairs of closely linked genes. The material is the same but in the second case we have pushed the analysis a little further. In like manner the rhesus alleles are the same but Fisher claims that they are linearly differentiated into three loci. This does not in any way disprove their allelism. Crossing over between Fisher's alleles, if it occurs at all, might be called mutation from one Wiener allele to another. The difference between close linkage and allelism may be compared with that between races and species. As two races evolve becoming more divergent they eventually become separate species; but the point at which they should be so called is arbitrary. Similarly the distinction may be made between close linkage and allelism purely as a matter of convenience.

Another statement with which I would take issue is that there are "no dominance relationships" between certain of the blood group alleles. Referring to the Landsteiner series Strandskov states, "In 1925 Bernstein pointed out three autosomal alleles with two of them dominant over the third but neither of the first two dominant over each other." Actually Bernstein stated, using the letter R (recessive) instead of O, "von denen A und B über R dominant sind, während der Heterozygot AB für sich unterscheidbar bleibt." That the heterozygote is phenotypically distinguished does not mean that dominance is lacking, although this error is frequently found in the textbooks. There is here a relationship of reciprocal dominance of complementary allelism with *L*A dominant both to *L*B and to *L*O with respect to antigen A, while *L*B is dominant over *L*A and *L*O with respect to antigen B. It is now also known that *L*O determines an antigen and acts in this respect as a dominant over both *L*A and *L*B.

The same principle of complementary allelism applies to the genes governing the M-N blood types and to the rhesus series. The genes are pleiotropic, each determining various traits,—the antigens being dominant, the antibodies recessive. Thus the rhesus blood type RhDdCcEe has the three Rh antigens D, C and E and the three Hr antigens d, c and e, but can form none of the antibodies against these because it is not homozygous for any one of these three pairs of alternative traits. There would be four different genotypes forming the blood type RhDdCcEe, namely *Rh*DCE/*Rh*dce, *Rh*DCe/*Rh*dcE, *Rh*DcE/*Rh*dCe and *Rh*Dce/*Rh*dCE. Although heterozygous for each of the three series of alternatives and having six of the possible antigens, these individuals would all be homozygous recessive for Du and for Cu, Cw and Cv and would therefore be able to form antibodies against any of the antigens determined by these gene elements.

The thirty possible rhesus alleles might give thirty different homozygotes each having three antigens against which these homozygotes would be unable to form antibodies. All of the heterozygotes, 435 in number of genotypes, would show complementary allelism by reciprocal dominance of one to three additional antigens, and they would be unable to form antibodies against these, but could form antibodies against any of the antigens that they lacked.

Finally I would like to modify and expand Strandskov's definition "A gene is what behaves as a unit in heredity" to read "A gene is what behaves as a unit in the meiosis of the heterozygote." We have no evidence that the elements of a gene stick together in the meiosis of the homozygote, that the chromonemal limits of a gene are in any way set except by comparison with an allele. It is also to be understood that the unity of a gene is not absolutely constant. It may mutate: its structure may be reorganized: there may be recombination between its elements and the elements of its allele. We may at some time find that the gene elements A and B have recombined giving the possibility of a homozygous AB blood type. This change might then be called a mutation constituting an additional allele, *L*AB, in the Landsteiner series.

Literature Cited

1. Bernstein, F. *Zeitschr. f. indukt. Abstamm. u. Vererbunsl.* 37: 237-270. 192
2. Strandskov, Herluf H. *Jour. Here* 39: 108-112. 1948.

JUN 27 1950